The New England
Journal of Medicine

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Volume 232 JUNE 7, 1945 Number 23

LYMPHOSARCOMA OF THE BOWEL IN CHILDHOOD*

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PRIMARY neoplasms of the large or small intestine are extremely rare in childhood as compared with those in adult life. Except for true adenomatous polyps, most intestinal tumors encountered in early life are sarcomatous. Lymphosarcoma is one of the more frequent types of this group, and it is the purpose of this paper to describe our experience with this tumor in children under twelve years of age, as well as a case of reticulum-cell sarcoma.

PATHOLOGY

Lymphosarcoma was separated from the general group of lymphoma in 1893 by Kundrat,[1] who described it as a highly malignant tumor of lymphoid tissue having a fairly uniform cell type. The tumor may arise in the lymph nodes, spleen, tonsils and the lymph follicles of the gastrointestinal tract, and apparently in many organs not usually thought of as containing significant amounts of lymphoid tissue. Extensive studies including reviews of the literature on this subject were reported in 1919 by Graves,[2] in 1932 by Ullman and Abeshouse,[3] in 1940 by Sugarbaker and Craver[4] and in 1942 by Stout.[5]

The controversial subject of the classification and inter-relations of the various types of lymphoma exceeds the scope of this paper. In this regard reference may be made to the work of Gall and Mallory,[6] Jackson,[7] Warren and Picena[8] and Krumbhaar.[9] The close relation between the various manifestations of neoplasia of the lymphocyte and its precursors seems to be quite well established. The confusion in the terminology applied to these tumors is widely known. Gall and Mallory[6] were unable to predict from histologic examination of lymph nodes involved by so-called "lymphocytoma," "lymphoblastoma" or "lymphosarcoma" whether there were accompanying leukemic blood changes. Certain pathologists apparently believe that the diseases designated by the terms "lymphatic leukemia," "aleukemic lymphatic leukemia," "lymphoblastoma," "lymphocytoma," "reticulum-cell sarcoma" and "lymphosarcoma" are variants of the same fundamental neoplasm.

Regardless of the inter-relation of these conditions, a malignant tumor of the lymphoid tissue may certainly appear as an original focus in the intestinal tract, and may remain temporarily local but ultimately invade and metastasize in a manner closely resembling that of carcinoma. The term "lymphosarcoma" may conveniently be applied to this neoplasm, despite certain theoretical objections.

The tumor arises in the lymphoid follicles of the submucosal layer of the bowel and infiltrates the remaining layers, although tending to remain intramural, and to extend through mucosa and serosa only as a relatively late change. Longitudinal and circumferential subserosal extension may cause diffuse thickening and dilatation of a segment of bowel, giving rise to the so-called "garden hose" appearance. The absence of a well-developed fibrous stroma as in carcinoma accounts for the rarity of the "napkin-ring" constriction of the bowel so frequent in the latter. Yellowish-white, subserosal plaquelike tumors, frequently umbilicated and frequently multiple (Fig. 1), represent a relatively early stage of the process. Since the mucosa is not primarily involved, large fungating masses completely enveloping the bowel may develop before intestinal obstruction is caused (Fig. 5). For the same reason, ulceration due to ischemic necrosis of the mucosa is a late result, in direct contrast to its early appearance in carcinoma. On palpation the lymphosarcomatous mass is softer, more friable and more rubbery than the hard mass of carcinoma.

Elaborate cytologic classifications of the subtypes of lymphoma have been developed by several pathologists.[6–8] For practical purposes two general groups are recognized — lymphosarcoma and reticulum-cell sarcoma. In the first group the type cell is the lymphocyte or lymphoblast. Both groups are characterized microscopically by destruction of normal

*Read by title at the annual meeting of the New England Surgical Society, Boston, September 20, 1944.
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nodal architecture, capsular invasion, numerous mitoses and predominance of one of the above cell types.

In our 5 cases of lymphoma arising primarily in the bowel, 2 were of the lymphocytic variety, 2 were lymphoblastic, and 1 was of the reticulum-cell group.

Clinical Aspects

In a recent ten-year study of histologically verified malignant tumors at the Children's Hospital, Farber[10] found 75 (21 per cent) belonging to the leukemia-lymphoma group. Of these 47 were classified as acute leukemia, 14 as Hodgkin's disease and 14 as lymphosarcoma. Five of these patients had primary tumors of the bowel with symptoms and signs that led to operative intervention. Data of these cases are summarized in Table 1. The youngest patient was four months of age and the oldest seven. All were boys. This is in keeping with the male predominance reported in the literature.

Table 1. *Summary of Data from Five Children with Lymphosarcoma of Bowel.*

Case No.	Age	Sex	Clinical Picture	Site of Tumor	Operation	X-Ray Therapy	Terminal Leukemia	Course
	Yr.							
1	6 4/12	M	Fecal impaction and chronic intussusception	Ascending colon	Ileotransverse colostomy (biopsy)	600 r	?	Died
2	7	M	Abdominal mass	Ascending colon	Laparotomy (biopsy)	None	No	Died
3	4/12	M	Acute intussusception	Terminal ileum	Reduction and resection, with anastomosis	None	Yes	Died
4	2 1/12	M	Melena and rectal mass	Rectum	Abdominoperineal resection	3000 r	No	No recurrence; well for 15 mo.
5	6 6/12	M	Fecal impaction and acute intussusception	Terminal ileum	Mikulicz resection	1350 r	Yes	Died

The general incidence of lymphosarcoma of the bowel in children may be further illustrated by reports from other clinics. Cave[11] in 1932 found only 1 case in a child among 15 patients observed during a twenty-one-year period at Roosevelt Hospital. McSwain and Beal,[12] reporting 20 cases of lymphosarcoma of the gastrointestinal tract treated at New York Hospital during the last nine years, encountered this tumor in 2 children. Donovan[13] states that only 1 child with enteric lymphosarcoma has been observed in the last fifteen years at the Babies Hospital in New York City.

Ullman and Abeshouse[3] believe that there is no absolutely characteristic clinical picture of this disease. Symptoms may have an insidious onset or may present themselves as an acute abdominal catastrophe. Obviously, the manifestations of disease are more or less dependent on the location of the tumor and the degree of intestinal obstruction it causes. The youngest patient in this series, a four-month-old baby (Case 3), had the classic symptoms of acute intussusception and at operation was found to have one of the ileoileal type whose leading point was an intramural lymphosarcoma. Two older patients (Cases 1 and 5) had increasing constipation for four and three months, respectively, leading to fecal impaction. One of these patients developed acute intestinal obstruction a few days after hospitalization and by barium enema was found to have an ileocolic intussusception (Fig. 4) associated with a large tumor of the terminal ileum. The other patient proved to have a chronic colocolic intussusception resulting from lymphosarcoma of the cecum and ascending colon. The presenting complaint in another patient (Case 2) was an essentially asymptomatic irregular abdominal mass filling the right lower quadrant of the abdomen. In the last patient (Case 4), bloody stools attracted attention to a lemon-sized intramural reticulum-cell sarcoma of the rectum (Fig. 2).

The peripheral blood showed no detectable abnormality in any of these 5 patients at the time of admission. Terminally, however, 2 of them developed the blood picture of lymphoblastic leukemia.

Case Reports

Case 1. W. T. (CH149544), a 6-year-old boy, was admitted in June, 1931, because of intermittent abdominal cramps of 3 months' duration. Four months before entry, appendectomy had been carried out at another hospital for alleged acute appendicitis. Irritability, fatigue and anorexia but no vomiting accompanied the pain. There was increasing constipation, and 10 days before entry a mass was noted in the epigastrium.

Physical examination on admission showed a well-developed and well-nourished boy in no acute distress. In the right upper quadrant of the abdomen there was a rounded, firm, nontender mass 7 cm. in diameter. Hard fecal lumps were palpable in the sigmoid and rectum. The remainder of the examination disclosed nothing remarkable. The urine, blood and stools were normal. After evacuation of the impacted feces by enemas, fluoroscopy of the colon showed a filling defect in the transverse colon suggesting intussusception. A diagnosis of chronic intussusception was made. Laparotomy revealed an irreducible intussusception of the ileocolic type that had passed just beyond the hepatic flexure. There were numerous large rubbery, yellow nodules in the omentum, which was adherent to the ileum and had been drawn into the intussuscipiens. The mesenteric nodes were large and firm. Biopsy was taken of the omental nodules, and a short-circuiting lateral anastomosis between proximal ileum and transverse colon was carried out.

Postoperatively, the patient did fairly well for a short period. After the pathologic diagnosis of lymphosarcoma had been made, it was decided not to do a second-stage resection of the chronic intussusception as planned but to give roentgen-ray therapy alone. Because of delayed wound healing only 600 r of x-ray radiation was given, following which the patient was discharged home on the 35th day, to return for heavy roentgen therapy when the wound was well healed. Because of parental difficulties, however, he did not return for admission but died at home 1 month after discharge. Repeated blood examinations showed no evidence of leukemia.

Microscopical study of an omental biopsy showed diffuse infiltration of omental fat by closely packed lymphocytic cells in no orderly arrangement. There were numerous mitoses. Diagnosis: lymphosarcoma.

CASE 2. E. C. (CH103886), a 7-year-old boy, entered the hospital in December, 1933, because of an abdominal mass noted 2 days previously. He had been well until 3 weeks before admission, when he became anorexic, listless and easily fatigued and complained of vague abdominal pains. The bowels were constipated. There was no vomiting. Two days before entry the family doctor was consulted, who sent the patient to the hospital because of a large mass in the right side of the abdomen.

Physical examination on entry showed a well-developed and well-nourished boy with a protuberant, distended abdomen. There was an enormous hard, nontender mass filling most of the right side of the abdomen, extending from the midline to the midaxillary line and from the costal border into the pelvis. Its lower limit was easily felt by rectum. Aside from additional signs of abdominal fluid the remainder of the examination was essentially normal. Urinalysis was negative. The red-cell count was 4,080,000 and the white-cell count 11,400, with 70 per cent neutrophils and a normal smear. Roentgenograms of the chest showed no metastases.

Laparotomy was carried out with a preoperative diagnosis of abdominal tumor. Opening of the abdomen revealed a grapefruit-sized, whitish-yellow tumor of the cecum and ascending colon. This was diffusely infiltrating and enveloped the large bowel, extending posteriorly to the abdominal wall and medially to the omentum. Several adjacent loops of small bowel were adherent to the mass. The mesenteric lymph nodes were enlarged, rubbery and hard. There was no hope of successful resection. A biopsy of the tumor was performed, and the abdomen was closed.

Postoperatively, the patient had a rapid downhill course. Although he did not become obstructed, he quickly developed marked cachexia. Delayed wound healing prevented early roentgen-ray therapy, and death occurred from terminal pneumonia on the 16th postoperative day. The peripheral blood showed no evidence of lymphatic leukemia.

Microscopical examination of the biopsy specimen showed closely packed masses of lymphoblasts with numerous mitotic figures. Diagnosis: lymphosarcoma.

CASE 3. F. R. (CH264439), a 4-month-old boy was admitted on July 29, 1942, with a 7-day history of irritability and refusal of feedings. In the 24 hours before entry he had vomited twice. The stools had been normal. The past history was noncontributory.

Physical examination on admission showed a well-developed and well-nourished baby crying as if with pain. The abdomen was slightly distended, but without tenderness, spasm or a palpable mass. The liver edge and the tip of the spleen were just palpable. The rest of the examination was essentially negative. The urine showed a ++ test for albumin but was otherwise negative. The red-cell count was 4,200,000 and the white-cell count 6000, with 71 per cent neutrophils, 22 per cent lymphocytes, 5 per cent monocytes, and 2 per cent eosinophils. The platelets were normal.

During the first 12 hours on the ward the patient refused feedings and vomited three times. He then began to develop intermittent episodes of abdominal pain with doubling of the knees on the abdomen. Fluoroscopy with a barium swallow revealed dilated loops of small bowel and fluid levels. A diagnosis of intussusception was made and laparotomy was performed. An enteric intussusception of the ileoileal type was present in the distal ileum. This was easily reduced, and the congested bowel quickly regained normal color. The cause of the intussusception was identified as two plaque-like, yellowish, intramural tumors of the intussusceptum measuring 2.5 by 1.5 cm. in diameter, respectively. These were situated on the antimesenteric border of the ileum 3 or 4 cm. apart. This segment of ileum was consequently resected (Fig. 1) and a lateral anastomosis was constructed.

FIGURE 1. *Photograph of a Surgically Excised Segment of Ileum Containing Two Intramural Lymphosarcomas That Had Caused Acute Intussusception (Case 3).*

Postoperatively the patient had a stormy course for 5 days, and then passed stools satisfactorily and remained afebrile. Repeated blood smears showed no evidence of lymphatic leukemia. No roentgen-ray therapy was given. The patient was discharged home in apparently good condition on the 18th day. He quickly became listless, irritable, anorexic and pale and developed a petechial rash over the extremities. He was readmitted to the Medical Service 10 days after discharge with a picture of advanced acute lymphoblastic leukemia. The liver and spleen were enlarged as were the cervical lymph nodes. The red-cell count was 2,520,000 and the hemoglobin 6.5 gm. The white-cell count was 76,000, with 4 per cent neutrophils, 3 per cent eosinophils, 4 per cent monocytes, 15 per cent mature lymphocytes and 74 per cent lymphoblasts. No platelets were seen on the smear. Therapy was omitted at the parents' request, and the infant died at home 5 days after the second hospital entry.

Microscopical examination of the intramural tumors of the excised ileum showed diffuse infiltration of bowel wall with masses of cells resembling lymphocytes and less numerous lymphoblasts. Mitoses were frequent. Diagnosis: lymphosarcoma.

CASE 4. J. B. (CH273465), a 26-month-old boy, was admitted on June 5, 1943, because of bloody stools of 25 days' duration. The past history was irrelevant. Except for diminished appetite and failure to gain during the month prior to admission there were no symptoms. Examination by the family physician had revealed a mass in the rectum. Proctoscopy and biopsy had been carried out in another hospital, followed by referral to this hospital with a diagnosis of malignant tumor of the rectum.

Physical examination on admission showed a pale, thin boy with a hard, smooth, lemon-sized tumor on the posterior rectal wall, extending to the brim of the pelvis. There were no other significant findings. Routine blood and urine examinations were normal. Roentgenograms of the skull, chest and skeletal system showed no evident metastases. into the lumen, except over a 2-cm. ulcerated area near the center of the mass (Fig. 2). Microscopical examination revealed a tumor consisting of large polygonal cells with abundant purplish cytoplasm and clear vesicular nuclei. Phagocytosis was prominent. Tumor cells were found in the blood vessels and lymphatics and diffusely infiltrated the entire rectal

FIGURE 2. *Photograph of a Resected Reticulum-Cell Sarcoma of the Rectum (Case 4).*

Consequently, a one-stage combined abdominoperineal resection of the rectum and lower sigmoid containing the neoplasm was carried out. The tumor lay in the wall of the rectosigmoid and was removed intact. Several large nodes in the mesosigmoid were excised. Postoperatively, the patient did well except for a troublesome urinary infection. He was given a wall, but did not extend through the serosa (Fig. 3). Reticulum fibers were demonstrated by Foot's stain. Diagnosis: reticulum-cell sarcoma.

CASE 5. G. H. (CH283174), a 6½-year-old boy, was admitted in May, 1944, with fecal impaction and a story of

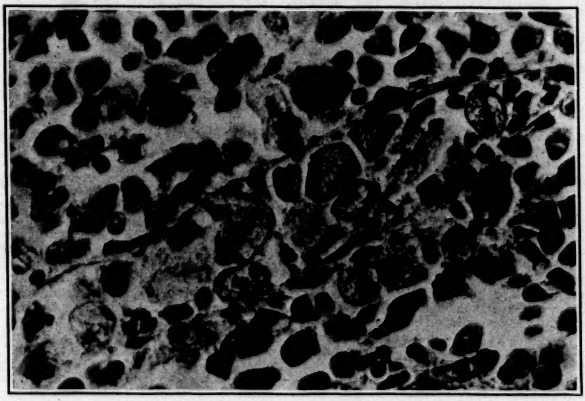

FIGURE 3. *Photomicrograph of a Reticulum-Cell Sarcoma of the Rectum (Case 4). Note the tumor cells in the lymphatic vessel.*

total of 3000 r of roentgen-ray therapy to the abdominal drainage area and was discharged home on the 60th day. He has remained in good health and has shown no evidence of recurrence of tumor.

The excised rectum and lower sigmoid contained a hard, spherical, yellowish intramural tumor measuring 7 by 4.6 by 4 cm. The rectal mucosa covered the tumor, which bulged progressive constipation following appendectomy at another hospital 4 months before entry. There had been no bowel movement in the 7 days prior to admission, and for 2 days the patient had had crampy epigastric pain. There had been no vomiting, anorexia, weight loss or melena.

Physical examination showed a thin boy with a protuberant, distended abdomen filled with hard, nontender movable

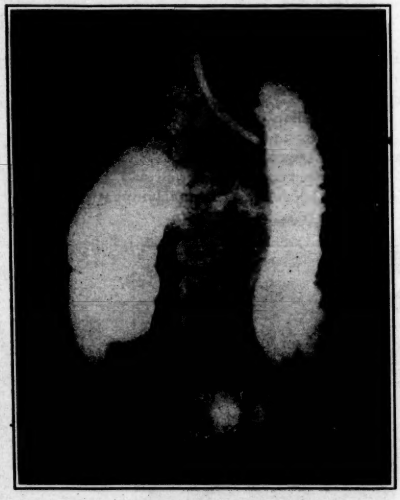

FIGURE 4. *Roentgenogram of a Barium Enema in a Patient (Case 5) with Lymphosarcoma of the Ileum.*

An ileocolic intussusception was partially reduced by the radiologist, with a residual filling defect in the cecum.

masses. The liver and spleen were not enlarged. The rectum contained concretelike fecal material. No other abnormalities were noted. Urinalysis was negative. The red-cell count was 3,800,000 and the hemoglobin 60 per cent. The white-cell count was 11,900, with 87 per cent neutrophils, 8 per cent lymphocytes and 5 per cent monocytes. No immature cells were noted, and the platelets appeared normal.

Cleansing enemas were given and copious fecal masses were evacuated, but there persisted a hard, grapefruit-sized mass in the lower midabdomen that could not be reached on rectal examination. On the 5th day signs of acute intestinal obstruction developed. A barium enema revealed displacement of the sigmoid to the left by a large, soft-tissue mass in the lower abdomen. In addition, there was an intussusception reaching the hepatic flexure; this was partially reduced by the roentgenologist (Fig. 4).

Laparotomy was immediately carried out and revealed a grapefruit-sized, hard, rubbery, yellowish-white tumor. The terminal ileum ran directly through the mass, which was situated 25 cm. from the ileocecal junction. The omentum was densely adherent to the anterior surface of the tumor, and the latter extended across to the sigmoid, which it had invaded. Four or five yellow plaquelike tumors measuring 4 to 6 cm. in diameter were present in the proximal ileum, jejunum and root of the mesentery. The terminal 15 to 20 cm. of the ileum was edematous and reddened, suggesting a spontaneously reduced intussusception. The large tumor of the ileum was freed from its attachments to the omentum and sigmoid, and a Mikulicz resection of the terminal ileum bearing the tumor was carried out (Fig. 5).

Postoperatively the patient went steadily downhill, rapidly developing malignant cachexia. Recurrent intestinal obstruction necessitated Miller–Abbott suction and a second laparotomy with lysis of adhesions. One thousand three hundred and fifty r of roentgen-ray therapy was given. During the 4th week lymphoblasts appeared in the blood smears. The spleen became enlarged, and the whole lower abdomen became filled with masses of tumor. There was a terminal leukopenia with diminished platelets and a rise in lymphoblasts to 16 per cent. The patient died on the 39th postoperative day.

The surgically excised tumor of ileum showed on microscopical examination masses of rapidly growing lymphoblasts exhibiting numerous mitoses (Fig. 6). Autopsy showed severe emaciation and purpuric hemorrhages in the skin and viscera, with massive extensions of tumor to the mesenteric and aortic nodes, several loops of the ileum and the sigmoid and bladder. Metastatic nodules were present in the kidneys, diaphragm, liver, lungs, testes and spleen. The bone marrow was heavily infiltrated. Diagnosis: lymphosarcoma, with generalized metastases.

FIGURE 5. *Photograph of a Resected Lymphosarcoma of Ileum That Had Extended to the Sigmoid and Root of the Mesentery (Case 5).*

Discussion

Preoperative recognition of lymphosarcoma of the intestinal tract in childhood offers many difficulties. In adults, bowel lesions of this type are usually confused with carcinoma until histologic examination clarifies the issue. The extraordinary rarity of carcinoma of the bowel in children is of considerable aid in differential diagnosis, and in all probability the practical consideration that a neoplasm of the intestinal tract in an infant or child is lymphosarcoma until proved otherwise is justifiable. The frequency of intussusception seems worthy of comment. This occurred in 3 of these 5 patients.

In our opinion a combination of radical surgical extirpation of the involved bowel and mesentery followed by heavy roentgen-ray therapy to the abdominal and mediastinal drainage areas offers the patient the optimal chance of survival, if, indeed, any such chance may be called optimal in this highly fatal disease. The mortality in this series was 80 per cent. The sole surviving patient has had no evidence of recurrence during the fifteen months since the abdominoperineal resection of the rectal tumor.

Summary

Four cases of lymphosarcoma and 1 case of reticulum-cell sarcoma of the bowel treated at the Children's Hospital during the last fifteen years are reported.

The pathologic and clinical aspects are discussed.

Radical surgical extirpation followed by roentgen-ray therapy seems to be the most logical treatment.

The mortality in this series was 80 per cent. Two patients developed terminal lymphatic leukemia.

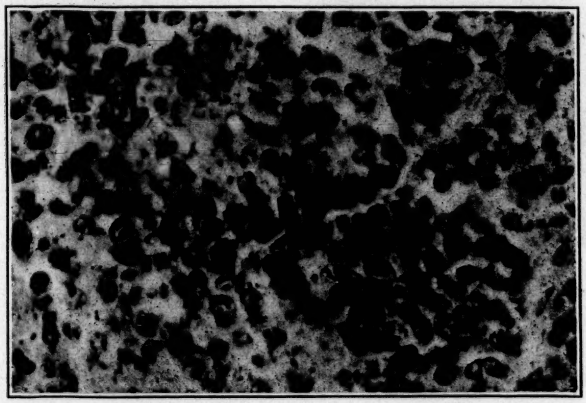

Figure 6. *Photomicrograph of a Lymphosarcoma of the Ileum (Case 5). Note the large lymphoblasts with numerous mitoses.*

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SHOCK IN ACUTE INFECTIONS

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THE recognition and treatment of shock have received much attention in the last few years, especially since the beginning of the war. Emphasis has been focused largely on the shock that occurs following surgical emergencies, such as injuries, hemorrhage and burns, and little attention has been directed toward shock in the course of acute infections. Since the occurrence of shock in association with infections is not infrequent, it is the purpose of this paper to stress the importance of the recognition of this syndrome and its vigorous treatment in combination with equally vigorous specific treatment of the infection.

Shock, regardless of its cause, is the result of a disparity between the circulating blood volume and the size of the vascular system. This may occur as a result of a loss of fluid from the circulation or an uncompensated increase in the size of the vascular bed. The latter factor is of most significance in the present study, although undoubtedly dehydration does play a part. Clinically this syndrome is manifested in the typical case by apathy, a cold, clammy skin, shallow respirations, a thready, rapid pulse and low blood pressure.[1–3] It has been the intent of recent surgical studies to point out the need for earlier diagnosis and thus earlier treatment of shock in order that the irreversible state toward which this condition tends to progress may be prevented.[1,4] In acute overwhelming infections, however, it is often impossible to anticipate the development of secondary shock, which frequently occurs precipitously. The peripheral vascular dilatation so often seen in acute infections is not to be confused with shock. The former is characterized by a flushed, warm skin, a bounding pulse, digital pulses and murmurs over the great vessels and is due to arteriolar dilatation, which is the customary response of the body to fever.[5]

The recognition of shock of the so-called "secondary type," or what may better be termed "cold shock," during the course of acute infection is not new. The weak heart sounds of a failing circulation in acute febrile illnesses were described in 1831 by Laennec,[6] but were erroneously attributed by him to heart failure. This concept of heart failure persisted in the medical literature, and it remained for Janeway[7] to show that the vascular collapse seen in the course of severe infections was due not to heart failure but to failure of the peripheral circulation with pooling of the blood in the capillaries. In his paper, he stressed vasomotor failure as the cause of death at the height of such infectious diseases as pneumonia, typhoid fever and septic fevers and referred to the experimental work of Romberg, Pässler et al.[8] The latter authors as early as 1899 had produced vascular collapse in rabbits by the intravenous injections of various bacteria, and had suggested the term "toxic shock" for the apparently similar vascular collapse occurring in acute infections. In 1930, Atchley,[9] in an exceedingly informative paper again brought out the importance of shock in medical conditions and stressed its early recognition and treatment, and in 1934, he and Loeb[10] reported studies along similar lines. The appearance of shock due to hemolytic streptococcus infections of postoperative wounds was emphasized by Stewart.[11] In all of Stewart's cases the state of shock was recognized and specific antishock therapy was instituted. The infection producing the shock, however, was not immediately recognized, and all the patients died in spite of adequate shock therapy in the absence of vigorous antibacterial therapy.

The following cases are typical of the therapeutic problems presented by three serious infections not infrequent in Army medical practice. We do not believe that these cases are comparable to those of Ebert and Stead,[12] whose patients were in the older age group and thus more prone to degenerative changes of the cardiovascular system.

CASE 1. A 37-year-old man was admitted to the hospital with a history of cough for several days and chilliness on the day preceding admission. Examination revealed nasal congestion and moderate pharyngeal injection. The white-cell count was 4000, with 58 per cent polymorphonuclear leukocytes and 42 per cent lymphocytes. The urine was free of albumin and sediment. Its specific gravity was 1.020. The oral temperature was 99.4°F. The course was benign until the afternoon of the 3rd hospital day, when the temperature rose to 103°F. At that time the patient's face was flushed and he vomited and had mild diarrhea. The vomiting continued, and at about 7:00 p.m. he developed a typical scarlet-fever rash.

The patient's general condition rapidly deteriorated. At 6:00 a.m. of the 4th day the rectal temperature was 105.4°F., the pulse 140, and the respirations 40 to 50 (Fig. 1). The white-cell count was 11,850, with 80 per cent polymorphonuclear leukocytes. Shortly he lapsed into a state of profound cold shock, the rectal temperature fell to 100°F., the extremities became cold and clammy, the pulse threadlike and barely perceptible and the lips cyanotic. The systolic blood pressure was 58; the diastolic pressure could not be determined. There was marked air hunger, and the patient had severe hiccoughing.

Energetic treatment was promptly begun. One hundred cubic centimeters of pooled human convalescent scarlet-fever serum was administered intravenously. The patient was placed in an oxygen tent, and an infusion of 1000 cubic centimeters of 5 per cent glucose in normal saline solution followed by 1000 cc. of plasma was administered during the first 24 hours. At the outset of the rather sudden collapse, he had been given caffeine. The hematocrit index prior to the giving of the plasma was 56 per cent and blood nonprotein nitrogen was 58 mg. per 100 cc. Intravenous fluids were continued, and the pulse gradually improved in quality and slowed so that it fell below 100 in 48 hours. The respirations returned to normal 24 hours after treatment of the scarlet fever and shock was instituted.

At 7:00 p.m. on the 4th day, the blood pressure began to rise and reached 70/54 at that time. On the following day it

was 90/68, and on the 6th day 110/80. Thereafter it remained within normal limits.

From the outset of the patient's acute collapse and for 72 hours thereafter he was anuric, and the blood nonprotein nitrogen rose to 130 mg. per 100 cc. Improvement in the renal function paralleled the rise in blood pressure, and the blood nonprotein nitrogen gradually dropped, becoming normal 6 days later. There was never any evidence of cardiac failure, and the patient was eventually discharged to duty.

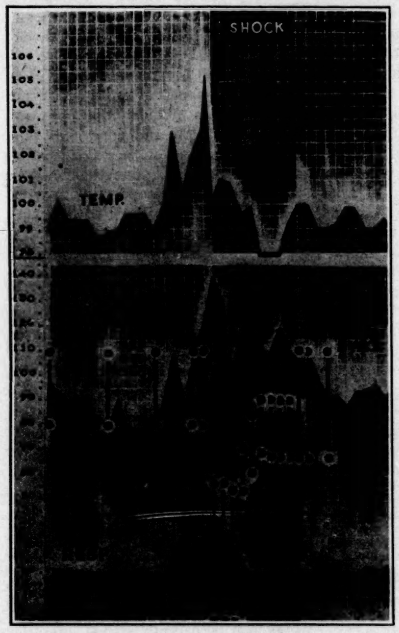

FIGURE 1. *Scarlet Fever (Case 1).*

In this and the two subsequent figures, each vertical subdivision represents a four-hour period. The temperature recordings were oral, except in the darkly shaded "shock" areas, when they were rectal.

CASE 2. A 37-year-old officer was admitted to the hospital complaining of generalized malaise, chilly sensations and a mildly sore throat. He had felt perfectly well until 2 hours prior to coming to the hospital, at which time he commenced to feel chilly and thought he was coming down with a "cold."

Physical examination showed an injected throat of moderate severity but was otherwise negative. The temperature was 102°F., the pulse 105, and the respirations 19. The white-cell count was 13,600, with 67 per cent polymorphonuclear leukocytes and 33 per cent lymphocytes. The urine was negative. A tentative diagnosis of pharyngitis was made and the patient was admitted to a respiratory ward.

Early the next day — 9 hours after admission — he experienced a severe chill and the temperature rose to 105°F. (Fig. 2). Physical examination was still essentially negative, although the patient's general appearance was quite toxic. Six hours later a profuse, blotchy, hemorrhagic skin rash commenced, breaking out over the entire trunk, extremities and face and even involving the mucous membranes of the mouth and pharynx. The pulse was imperceptible, and the systolic blood pressure was 65. Respirations were shallow at 40, and the rectal temperature was 101°F. The white-cell count was 24,000, with 95 per cent polymorphonuclear leukocytes. A diagnosis of septicemia with severe secondary shock was made, and blood cultures were taken that later grew Type 2*a* meningococci. The spinal fluid was clear, and no organisms could be cultured from it.

Antibacterial therapy was immediately instituted and consisted of 5 gm. of sodium sulfadiazine in 100 cc. of sterile water intravenously and 4 gm. of sulfadiazine by mouth, followed by 1 gm. of sulfadiazine orally every 4 hours. Sodium bicarbonate was administered orally — 3 gm. per gram of sulfadiazine. Concomitantly with the institution of chemotherapy, efforts were directed to combat the severe and apparently progressive shock that the patient manifested. Two thousand cc. of 5 per cent glucose in normal saline solution, 1000 cc. of 10 per cent glucose in distilled water and 500 cc. of plasma were administered daily for the first 3 days. In addition, the patient was urged to take water and fruit juices by mouth. Five milligrams of cortate was given intramuscularly every 4 hours, and the foot of the bed was elevated on shock blocks.

On this regimen a blood pressure of 60/40 to 80/50 was maintained for the first 2 days. One hundred and five cubic centimeters of urine was passed the first day. On the second day, however, a total of 1500 cc. of urine was passed, and from then on the urinary output remained satisfactory. The blood pressure rose to 100/60 on the afternoon of the 3rd day and over the next 3 days gradually returned to a normal level (120/80). Intravenous fluids were abandoned after the 3rd day. Chemotherapy was stopped on the 12th day, and convalescence was uneventful.

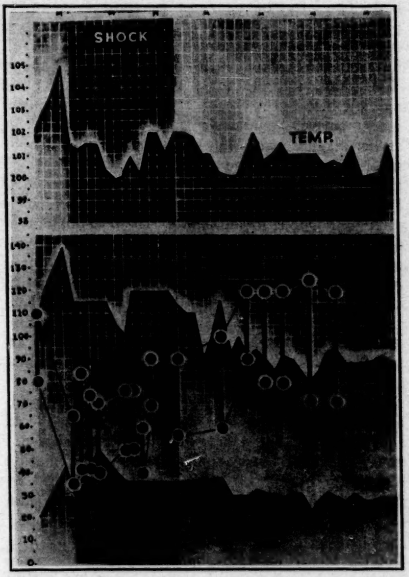

FIGURE 2. *Meningococcemia (Case 2).*

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CASE 3. A 25-year-old soldier was admitted to the hospital because of a cough and moderate pain in the right chest of 4 days' duration. His previous health had been excellent. On admission he was moderately ill.

Physical examination revealed rales over the right lower lobe, and an x-ray film taken the day of admission showed an infiltration in the same area. The temperature was 102°F. and the white-cell count 10,400, with 69 per cent polymorphonuclear leukocytes. The urine was normal. A diagnosis of primary atypical pneumonia was made, and the patient received symptomatic therapy.

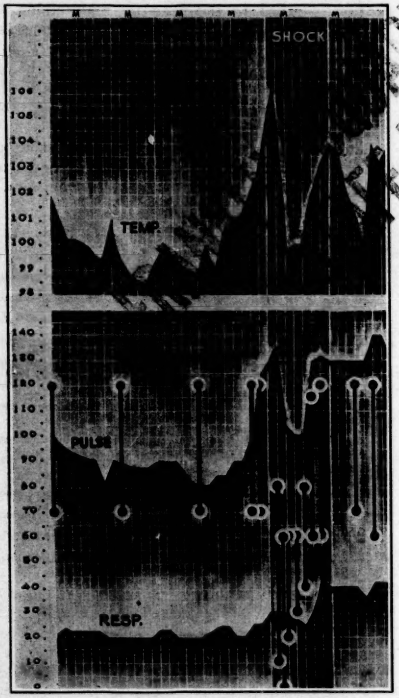

FIGURE 3. *Pneumococcus (Type 3) Pneumonia (Case 3).*

During the first 4 hospital days his course was uneventful and he appeared to be recovering. On the 5th day the rectal temperature rose to 106.2°F. (Fig. 3) in 4 hours. The respirations became labored. The blood pressure dropped precipitously from 120/70 to 70/0 and later to 60/0, and shortly thereafter the rectal temperature decreased to 100°F. The skin was cold, and the pulse was imperceptible. The patient was extremely restless, and there was cyanosis of all the extremities.

Examination of the chest at that time revealed signs of frank consolidation at the right base, although the white-cell count was still only 12,000, with 74 per cent polymorphonuclear leukocytes. The sputum contained Type 3 pneumococci. Blood cultures were negative. It was believed that the patient had developed a severe bacterial pneumonia superimposed on the original infection and that the state of profound shock was secondary to the acute infection. He was given 2000 cc of 5 per cent glucose in normal saline solution and 500 cc. of plasma over the next 12 hours. Simultaneously with the above measures, he was placed in an oxygen tent and started on sulfadiazine therapy. Five grams of sodium sulfadiazine was given intravenously, followed by 1 gm. of sulfadiazine orally every 4 hours. Sodium bicarbonate was given orally, 3 gm. per gram of sulfadiazine.

The blood pressure gradually rose to 120/70 over the next 24 hours, and with its return to normal all signs of shock disappeared and the urinary output improved. Because the patient continued to run a febrile course, however, he was given 200,000 units of Type 3 antipneumococcus serum in addition to the sulfadiazine. The spiking fever persisted, and on the 8th day signs of fluid developed in the right chest. A right-sided thoracentesis revealed purulent fluid containing pneumococci. On the 20th day closed-tube drainage of the right chest was instituted, and from then on convalescence was uneventful.

Figures 1, 2 and 3, which record the temperature, pulse, respirations and blood pressure determined at four-hour intervals in each case, emphasize several points of similarity that are readily noted graphically and that may warn clinicians of impending events. There was an abrupt rise in temperature to a high level shortly preceding the overwhelming spread of the infection. This was followed by an equally abrupt fall in temperature, although the pulse and respirations did not correspondingly drop. Oliguria was present during the period of hypotension. Each patient had been examined prior to the onset of shock and clinically presented evidence of an essentially negative cardiovascular system and appeared to be in good fluid balance.

The mechanism producing the shock in all 3 cases is probably due to the sudden invasion of the vascular system by bacteria or bacterial toxins or both that paralyzes and dilates the capillary bed. A pooling of blood occurs in the dilated capillaries, with a concomitant reduction in the venous return to the heart and in the cardiac output. Romberg et al.[8] demonstrated in 1899 that the injection of bacteria and bacterial products intravenously could produce vascular collapse, and other investigators[10,13] have confirmed their findings.

The first case presented in this study was one of severely toxic scarlet fever. Presumably in this type of case one is dealing with a true bacterial exotoxin that is absorbed into the blood stream. By the intravenous administration of pooled convalescent scarlet-fever serum the temperature and some of the toxic manifestations were controlled. However, the tone of the vascular system did not return until the fourth day, and during that time intravenous fluid and plasma were necessary to maintain the blood pressure at a critical level. The disparity between the circulating blood volume and the size of the vascular bed was further aggravated by the loss of fluid through vomiting and diarrhea.

The second case was one of a fulminating meningococcemia, with a massive invasion of the blood stream by bacteria and bacterial products. As is

well known, many of these patients die in severe shock before the infection can be controlled by chemotherapy. In this case again, it was possible by the use of intravenous fluid and plasma to maintain the circulating blood volume and blood pressure at a critical level until the infection could be controlled by sulfadiazine.

In the third case, one of Type 3 pneumococcus pneumonia, although blood cultures failed to demonstrate a septicemia it was thought that the severe state of peripheral vascular collapse was due to an invasion of the blood stream both by bacteria and by bacterial products. Here again, while the infection was being treated energetically with sulfadiazine and Type 3 anti-serum, the circulating blood volume was maintained by intravenous plasma and fluids.

The institution of specific measures to combat the infection is of primary importance, since they are directed at the cause of the shock state. It is our contention, however, that death in the early stages of acute fulminating infections may occur as a result of secondary shock; furthermore, if vigorous anti-shock therapy directed to maintain an adequate circulating blood volume is resorted to, as in the cases cited above, many patients may be maintained until specific serum or chemotherapy or both have been able to control the infection.

It is considered advisable to give warning that overly enthusiastic use of procedures designated to restore inadequate circulating blood volume in patients with certain types of acute pneumonia, particularly interstitial pneumonia, so frequently seen in young children, may result in an increase in exudation in the lungs and harm to the patient.

Summary

Shock during the course of acute infection is discussed, and its importance as a preventable cause of death is stressed.

In the treatment of shock in acute infections, attention is called to the need for maintaining an adequate circulating blood volume by the use of intravenous plasma and fluids while the infection is being vigorously treated by appropriate chemotherapy or serotherapy. The use of oxygen in the face of anoxemia and other supportive measures are not to be neglected.

Three case reports that illustrate acute infections not infrequently seen in medical practice and that presented serious states of shock are given in detail, with their treatment.

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MEDICAL PROGRESS

OPHTHALMOLOGY

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THE following ophthalmologic subjects have been selected as being of general interest. Some of them are not new, but because of recently revived interest in them, they were thought to deserve review.

RELATION OF GERMAN MEASLES TO CONGENITAL CATARACTS

One of the most interesting recent developments in the field of ophthalmology has been the recognition of the fact that congenital cataracts can be caused by German measles occurring in the mother early in pregnancy. Gregg,[1] of Sydney, was the first to point out this relation, and in 1941 he reported a series of cases to the Ophthalmological Society of Australia. He stated that in the summer of 1940 there had been an epidemic of unusually severe rubella in southeastern Australia. The following year there occurred a great increase in the incidence of congenital cataracts among the newborn of Sydney and other neighboring cities. Gregg reported 78 cases of cataracts, 20 of which were seen by him and the remaining 58 by colleagues and reported to him. In all but 10 cases, a history of rubella during early pregnancy was obtained. The cataracts were bilateral in 62 cases and unilateral in 16. Microphthalmia was present in 10 of the unilateral cases.

According to Gregg, these cataracts were of a special type, being dense white with all layers of the lens involved. Most of the babies were small and ill nourished and presented definite feeding problems. Forty-four of them had congenital heart disease. Fifteen deaths were recorded. Autopsy in 3 cases revealed patency of the ductus arteriosus.

An interesting feature was the reported intolerance to atropine exhibited by all these infants. When 1 or 2 drops of a 0.5 per cent solution of this drug were used to dilate the pupils, considerable constitutional disturbance took place, consisting of flushing of the skin, pyrexia and disturbed digestion.

Gregg believed that there was more than a casual relation between the attack of rubella and the occurrence of the congenital cataracts. In 35 cases in which accurate records were available, the affected baby was the first child in 26 cases, the second child in 3, and the third, fourth, fifth, sixth, eighth and tenth child in the remaining. Other children in the families showed no signs of cataracts, nor did the parents.

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Gregg raised the question whether the causative factor was really German measles because of the severe epidemic of sore throat that was sweeping the country concurrently with the rubella, most of the mothers having had tonsillitis at the time of their illness. He suggested that the rash diagnosed as German measles might have been a toxic erythema accompanying a streptococcal throat infection.

Naturally this report stimulated great interest throughout Australia, and under the direction of the National Health and Medical Research Council a group was appointed to determine the following points: whether the disease of the mothers was rubella or some illness simulating it, the period of pregnancy in which the disease produced congenital defects, the full extent of these defects and whether these defects could have been produced by other infectious diseases during pregnancy. This extensive investigation was conducted by Swan, Tostevin, More, Mayo and Black.[2] All the physicians in certain sections were circularized, and all pregnant women with exanthematous diseases were registered. All new cases of congenital cataracts in infants were investigated, and the history of each mother was carefully gone over for evidence of prenatal illness. The results of this study were published in 1943 and showed that of 61 infants examined, 36 had congenital defects. Forty-nine of the mothers had had rubella during pregnancy, 9 had had morbilli and 2 mumps; 4 denied knowledge of any disease. Of the 49 mothers with rubella, 31 gave birth to infants with congenital defects. Of these, 13 had cataracts, in 10 of whom they were bilateral, 1 had buphthalmos, 7 were deaf mutes, and 17 had cardiac anomalies. Some degree of microcephaly was apparent in all. All but 2 of the mothers of the infants with congenital defects had contracted rubella during the first three months of pregnancy.

It was the conclusion of the investigators that there was no valid evidence that the epidemic of 1940 was other than rubella and that the predominating organism in the concurrent epidemic of sore throat was *Haemophilus influenzae*. Furthermore, they stated that subsequently, when there was no epidemic of sore throat, congenital cataracts were found in the offspring of mothers who had had German measles.

In a subsequent communication, the same authors[3] reported 12 additional cases of rubella occurring during early pregnancy, eleven of the infants showed congenital defects. These included 2 cases of cataracts, 5 of deaf mutism and 4 of heart disease.

If these two series are combined, it is seen that of 61 mothers who had rubella during pregnancy, 42 gave birth to children with congenital defects and of these 15 had cataracts. In analyzing the data, the investigators concluded that if a woman contracts German measles during the first two months of pregnancy, the chances of a congenitally defective infant are practically certain. If the disease occurs during the third month, about half the infants will be affected.

During the past year several American reports have corroborated Gregg's original findings. Thus, Reese[4] reports 3 cases of dense white cataracts and congenital heart defects in infants whose mothers had been treated for rubella during the first month of pregnancy. Rones[5] reports 3 maternal cases of rubella and 1 of morbilli. In 2 of these cases the exanthema had occurred in the second month of pregnancy and the infants were born with bilateral cataracts; 1 of them had congenital heart disease. In the other 2 cases the disturbance took place in the third month and the infants were born with congenital glaucoma.

Erickson[6] reports 9 cases of congenital cataracts in infants born of mothers who had had rubella during the first two and a half months of pregnancy. All but 2 of these showed heart defects in addition to the cataracts.

It is known that important developmental changes take place in ocular and cardiac tissues of the embryo during the first three months of pregnancy.[7] It is also known that embryonic tissues are especially susceptible to virus infections. Goodpasture[8] has pointed out the greater susceptibility of chick embryo, as compared with the hen, to a wide variety of infections inoculated in the chorioallantoic membrane. It is not improbable that the human embryo possesses the same increased susceptibility, so that infections such as German measles, which cause only slight or no damage to adult tissues, result in profound changes to embryonic cells.

With formation of the placenta at the end of the third month of pregnancy, there is apparently a reduction in the penetrability of the barrier between mother and fetus. This may in part explain the decreased incidence of congenital defects when the infections occur in the later months of pregnancy.

Little is known concerning the exact mechanism of cataract formation. Swan and his co-workers[2] suggest interference with the nutrition of the lens by action of the agent on the hyaloid artery. It seems more probable, however, that the lens is directly affected.

This whole subject brings up the question of whether abortion is justified if a woman contracts rubella during the first three months of pregnancy. Certainly it is desirable that girls be exposed to this disease before marriageable age. There are, of course, practical difficulties in this procedure.

Eye Exercises for Improving Vision

The question of eye exercises to improve visual acuity is one that crops up perennially and has for years been the subject of bitter debate. The Bates method,[9] originally brought forth in 1920, has recently been repopularized by Huxley's[10] book *The Art of Seeing*.

Bates, a New York physician, claimed that accommodation was due not to an increase in the convexity of the lens, as is generally believed, but to a change in the shape of the eyeball brought about by the action of the superior and inferior oblique muscles. He believed that all refractive errors were due to the unbalanced pull of these muscles. The animal experiments on which he based this theory were open to certain criticisms. Also, his claim that the cure of all refractive errors lies in so-called "dynamic relaxation," which can be brought about by certain exercises, was not accepted by the medical profession. The method was employed by many cultists, and after enjoying several years of popularity with the public gradually fell into disrepute, only to have a renaissance in recent years when men were being rejected for military service because of eye defects. Although the majority of orthodox ophthalmologists[11–15] decry such antics as "palming, swinging the eyes, sunning, thinking of black velvet and so forth" as pure nonsense, one must nevertheless admit that there are a certain number of people who, by these methods, do achieve improvement in their visual acuity without the aid of glasses. To the simple neurotic such a system may be expected to give aid and comfort. It is also understandable that it might benefit borderline cases of myopia, since many patients are not really myopic but, owing to excessive accommodation, have brought the focal point of the fixated object anterior to the retina, thus producing an artificial myopia. Learning to relax their accommodation, by whatever means employed, results in their being able to see normally without glasses. Lancaster,[16] who has approached the subject with an unbiased mind, illustrates this point with the story of a young man who came to him asking whether there was any chance of his passing the 20/20 visual test. His vision with his weak myopic lenses was 20/15 but without them was only 20/30. He was given +1.00 spheres to wear constantly for three days. He then read 20/15 without glasses. This man was not a true myope; he had simply learned to relax his accommodation by wearing convex lenses for a while. The same results could probably have been obtained by the Bates exercises.

Even in true myopes, going without glasses for a few days and learning to relax often results in some visual improvement on the test chart. It is not that the refraction has changed essentially, but that the patient has learned to interpret blurred objects more or less correctly through practice. This point has been emphasized by Lancaster.

Post[17] has collected the records of 57 cases of myopia in which the vision was definitely improved by one or another type of exercise. One of the most remarkable of these cases was that of a twenty-four-year-old man whose vision had become progressively worse through the years, it being 20/200 in each eye corrected to 20/20 with about —2.00 spheres and small cylinders. These tests had been made under a homatropine cycloplegia. Being rejected for military service, he took so-called "muscle exercises" for twenty-four lesson periods, at the end of which he was able to see 20/20 with each eye without glasses. Homatropine refraction then showed the same error as he had had before taking the exercises! In this case, care had been taken to prevent memorizing the test letters. The same ophthalmologist examined the patient on both occasions. Since his refractive error under the cycloplegia had not changed, his great improvement must have been due to learning how to use his visual powers to the utmost advantage by practice and repetition.

According to Lancaster, "seeing is only partly a matter of the image on the retina but is, in still larger part, a matter of cerebral synthesis in which memories play a principal role. By repetition one builds up a substratum of memories very useful for the interpretation of sensations and facilitates the syntheses which are the major part of seeing." Experience has shown that, although some patients are improved by these methods, the improvement is usually not lasting. Many patients state that they did not have time to keep up their exercises and eventually went back to wearing glasses. The dangers of the unsupervised use of these exercises in cases of detached retina and glaucoma have been pointed out by Duke-Elder.[18]

Men rejected for military service because of color blindness often apply for treatment to cure their defect. A number of optometrists have claimed that they are able to help these persons. The treatment consists in fixation of colored lights, the sorting of colored beads and the use of filters in viewing the test plates. By constant repetition the patients learn to interpret their sensations more efficiently, and undoubtedly a number of them have been able to pass the test. Subsequent reports from the Army and Navy[19] indicate, however, that the improvement in such cases has not lasted, patients later on having fallen down badly in critical situations that required accurate color perception.

According to Bridgman and Hofstetter,[20] two optometrists at Ohio State University, the alleged improvement in color vision after this type of training is due to learning made possible after repeated testing. They illustrate this by the report of a color-blind subject who at his first examination scored only 15 out of 46 plates. He was merely told each time that he was wrong but was not told the correct answer. On the second, third, and fourth testings, his scores were 27, 35 and 43 correct. These authors concluded that the color-filter treatment is not only ineffective in curing color blindness but is, in fact, dangerous, since it leads persons into the acceptance of responsibilities that they will be unable to fulfill.

It must be kept in mind that there are various degrees of color blindness and that in mild cases repeated testing leads to proficiency in correctly naming the test plates. When the scene is changed, however, such persons are still found to be "color weak." This is borne out by some recent unpublished work by Gallagher[21] and his associates. Forty-five students were selected as being color weak from their responses to the American Optical Company pseudoisochromatic test plates[22] and were given training on these plates until all could pass the test perfectly. They were then tested on the Ishihara plates,[23] which are quite similar, but only 6 of them made perfect responses on this test, which was unfamiliar to them. Several months later, 15 of them were retested on the American Optical Company plates and a sharp decrease in their ability again to name these plates correctly was noted. Even when these 45 students were at the peak of their proficiency in passing the American Optical Company test, they were unable to pass the color-desaturation test. In this test various saturations of red and green light are thrown on a screen in random order and the subject is asked to say whether the color is red or green. Gallagher and his associates conclude that there is no evidence that color training improves the ability of a color-weak subject to discriminate between colors except in the situation in which the training is given.

Effect of Fluorescent Lighting on Eyes

The fluorescent lamp has been an important development in electric lighting. Since its introduction some years ago, however, there have been many complaints that it is fatiguing to the eyes. Physicians are often asked by their patients whether it is actually harmful. It may be helpful, therefore, to review the evidence on this question.

The fluorescent lamp is a mercury-vapor lamp giving off chiefly ultraviolet radiations within a tube. The inside of the tube is coated with certain substances that fluoresce under this radiation. No ultraviolet light reaches the eye, as is generally believed by the laity. The lamp has the advantage of producing three times as much light per watt as the regular incandescent lamps. Another advantage is the relatively low degree of radiant heat given off when high levels of illumination are used, daylight fluorescent lamps giving forth only one quarter as much heat as an incandescent lamp of equivalent light output.[24] Furthermore, the comparative low brightness per square inch of glass surface and the long form of the tubes make it possible for the

fixtures to be hung at low levels with an even distribution of light.

Fluorescent lamps operate on alternating current, usually 60 cycles per second. With each change in the direction of the current there is a slight pause during which no current is flowing. This pause is almost infinitesimal, but during it the lamp becomes less bright. Under most circumstances this makes no difference. If, however, the eye is moving rapidly over a wide area, a series of successive images fall on different points of the retina and a series of separate sensations are observed, since each one persists after the next one begins. The same effect can be obtained if the eye is stationary but the object moves rapidly across the field. This flicker may give rise to disagreeable sensations under the above-mentioned conditions, but if only a small stationary area is being viewed, the successive images fall on the same position of the retina and a continuous or fused picture is produced.[25] This stroboscopic or flicker effect can be eliminated by combining tubes in pairs so that the maximum output of one coincides with the minimum output of the other. Indeed, most modern installations are of this type.

The whole question of fatigability under fluorescent light has been investigated by Luckiesh and Moss.[26] They base their conclusions on the results obtained by comparing visual performance under fluorescent light with equivalent illumination from tungsten-filament lamps. After testing a number of subjects they found no difference in visual acuity when either of these two types of illumination was used. The fluctuation in the light output of a daylight fluorescent lamp did not cause ocular fatigue as measured by the rate of blinking[27] during reading. This is not surprising since the flicker of the fluorescent lamp is well above the critical flicker frequency of the human eye and should not be bothersome if the eyes are fixed on a small area. The fixational pauses[28] that occur during reading were electrically recorded and were not found to be prolonged under fluorescent light.

From the above experiments Luckiesh and Moss concluded that there is no basis for the oft-heard criticism that fluorescent light fatigues the eyes. Although the criteria they used may be open to some criticism as not truly determining ocular fatigue, nevertheless no one has been able to bring forth any valid evidence that fluorescent light in ordinary office use is either harmful or tiring. If care is taken in installing the fixtures to minimize glare and to provide an adequate and even distribution of light, this type of illumination should be just as satisfactory as any other type now in use.[29, 30]

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CASE 31231

PRESENTATION OF CASE

A sixty-three-year-old business man was admitted to the hospital because of fever, dyspnea, inability to urinate and exacerbation of a chronic scrotal infection.

For several years he had had nocturia, frequency and hesitancy. These symptoms progressed, and two years before admission he had a transurethral prostatic resection in a community hospital. On the tenth postoperative day he developed a right epididymitis, which progressed to abscess formation and necessitated a right epididymo-orchectomy. Since that time he had had a draining sinus at the upper angle of the wound. The discharge was offensive and the sinus refractory to local treatment. The patient continued to work despite moderate frequency, a draining sinus and occasional bouts of left scrotal pain and swelling. Seven months before admission he experienced some nausea and abdominal discomfort. He went to another hospital where he was found to have anemia, with a red-cell count of approximately 1,000,000; the spleen was enlarged, extending to the level of the umbilicus. For several months he was treated for anemia. Six weeks before admission he was given three x-ray treatments to the spleen, which allegedly caused some decrease in size and was followed by a rise of the red-cell count. His appetite improved, and he gained weight. Since the operation two years before entry he had been taking 0.1 gm. of digitalis daily. Ten days before admission he experienced malaise and dyspnea and felt vaguely ill. A physician advised bed rest. Shortly after this the patient found that he could not urinate. After several catheterizations he developed pain in and swelling of the left scrotum. The temperature rose to 104°F., and after nine days on sulfadiazine therapy (6 gm. a day, with sodium bicarbonate) he became oliguric and was brought to the hospital. The dyspnea and moderately severe orthopnea, requiring two or three pillows, had continued. A cough that had been present and nonproductive for several years had, during the past few weeks, become productive of yellow, tenacious sputum, especially pronounced on change of position. He had had no chest pain, palpitation or swelling of the ankles.

Physical examination revealed a well-developed, well-nourished man with dry, hot and flushed skin. The throat was dry and red. The neck veins were distended and distinctly pulsating. The chest was emphysematous and showed limited motion. There were rales in both bases posteriorly and in the axillary regions. The diaphragm was low on both sides and showed limited motion. The heart was enlarged to just beyond the left midclavicular line. The sounds were distant. An inconstant apical gallop rhythm developed and persisted. No murmur or thrill was noted. The aortic second sound was greater than the pulmonic. The abdomen was flaccid; the liver was nontender and extended two fingerbreadths below the right costal margin. The spleen extended five or six fingerbreadths below the left costal margin and almost to the midline. There was distinct bilateral costovertebral tenderness, but that on the right side disappeared in a day. There was no fluid, and no other masses were felt. The right scrotum was empty and retracted. There was a small draining sinus at the upper angle of the orchectomy scar. The left testis was tender, and the left epididymis was enlarged, tender and hard, with an overlying warm, red skin. A slight purulent discharge came from the urethra. The penis was normal. Rectal examination revealed little prostatic tissue and no hemorrhoids. There were varicose veins of the legs and mild pitting edema of the lower extremities, sacrum and back. The legs were symmetrical, and the pulsations of the dorsalis pedis arteries were good.

The temperature was 104°F., the pulse 120, and the respirations 50. The blood pressure was 110 systolic, 60 diastolic.

Examination of the blood showed a red-cell count of 2,900,000, with 9.7 gm. of hemoglobin. The red cells showed quite marked anisocytosis and poikilocytosis. The white-cell count was 6400, with 89 per cent neutrophils; there were 2 per cent band forms and 1 per cent myeloblasts. A voided urine specimen was cloudy, amber and acid in reaction. The specific gravity was 1.006. There was a ++ test for albumin, and the sediment contained 3 to 5 granular casts per high-power field, as well as numerous white cells and crystals resembling sulfadiazine; no red cells were seen. The sulfadiazine level of the blood was 2.9 mg. per 100 cc. The serum nonprotein nitrogen was 85 mg. per 100 cc., and the protein 5.8 gm. The albumin-globulin ratio was 1.2. The carbon dioxide and chloride were normal. A smear of the discharge from the scrotal sinus showed no tubercle bacilli, but the culture yielded a moderate number of colonies of *Staphylococcus albus* and a few of a beta-hemolytic streptococcus. The urine culture showed moderate growth of a beta-hemolytic streptococcus and a few colonies of an alpha-hemolytic streptococcus. A Congo red

*On leave of absence.

test showed 76 per cent of the dye remaining in the blood in one hour. The Hinton test was negative.

X-ray examination of the chest showed diffusely increased vascular markings throughout both lung fields, normal hilar shadows and a smooth diaphragm, with no evidence of fluid in the costophrenic sinus. The respiratory rate was rapid, and the diaphragmatic excursion quite limited. The heart was not grossly enlarged, and the cardiothoracic ratio was 13.9:31 cm. The aorta was somewhat tortuous, with some calcification of the arch. One electrocardiogram on the third day showed auricular fibrillation with almost ventricular rhythm at the rate of 90; there was slight left-axis deviation, with sagging ST_1 and ST_2 and low upright T_1 and T_2.

No sulfadiazine or salt was given, and the patient was fully digitalized at once. The oral fluid intake was about 2500 cc., and the output about 1750 cc., with a slight parallel rise in these values during the first week. By lysis, the temperature became normal on the fourth day, and the pulse and respiratory curves followed. These remained normal for three days and then began to rise again. The nonprotein nitrogen rose to 135 mg. per 100 cc., and two days later, on the eighth hospital day, the patient expired.

Differential Diagnosis

Dr. Earle M. Chapman: It seems evident that this man suffered from chronic bacterial infection, especially of the scrotum, from which area *Staph. albus* and beta-hemolytic streptococci were cultured. The urine was likewise contaminated.

The chronic nonproductive cough that he had had for years became productive of purulent sputum on change of position during his final illness, and then rales were discovered at the lung bases. These facts justify a clinical suspicion of bronchiectasis. Unfortunately the x-ray studies do not help us to establish such a diagnosis.

With this background of chronic sepsis we are next faced with explaining a man's death in renal failure — a rather unusual form of renal failure that was further complicated by the use of two potent and often toxic drugs — digitalis and sulfadiazine.

There seems little question that 54 gm. of sulfadiazine in nine days is excessive, and the cessation of urine flow toward the end of his illness favors a specific type of damage to the kidney. On hospital entry, however, only 2.9 mg. of the drug was found in the blood stream, and no red cells in the urine. These two facts do not support my first suspicion that his kidneys might have shown the granulomatous changes of sulfadiazine intoxication.

As for the digitalis, a person who has taken 0.1 gm. daily for two years, and has nausea and abdominal discomfort seven months before entry, and is then given more of the drug on hospital entry is apt to become sick. The electrocardiographic changes, especially the sagging of the ST segments, are probable digitalis effects. How can the pathologist help us at autopsy in determining whether or not a person has been toxic from digitalis? I raised this question at the autopsy table recently and received little help.

The next thing to discuss is this man's death in renal failure. What type of kidneys should we expect to find? I am going to commit myself now to that unusual form called the "contracted amyloid kidney" — the kidney that in gross often has the appearance of a chronic glomerulonephritis. With this background of sepsis, allegedly nontuberculous, there might have been the condition favoring the release of protein complexes, with amyloid degeneration in the walls of the blood vessels in the kidneys and spleen.

I now wish to call your attention to the clinical features of renal amyloidosis in this man's record. The patient always had polyuria, — not influenced by transurethral resection, — and even toward the end he excreted a urine of low gravity and with the surprising daily volume of 1750 cc. He also had an anemia, chronic and resistant to treatment and yet not disabling; next, a normal-sized heart and a normal blood pressure — hypertension in renal amyloidosis is indeed rare; next there was a rather small amount of albumin in the urine, a low albumin-globulin ratio and, finally, a gradually rising serum nonprotein nitrogen.

Balanced against all this clinical evidence is one miserable laboratory test — the Congo red test. They tell us that it showed 76 per cent of the dye remaining in the blood in an hour. When Bennhold[1] described this test in 1923, he claimed that normally less than 30 per cent disappeared from the blood in an hour and that in amyloid disease 40 to 100 per cent leaves the blood in an hour. It has been found by others, however, that 40 to 60 per cent may remain in the blood in cases of amyloid disease and in other types of nephrosis, so that it is not always of practical value in determining the type of nephrosis. Bennhold did not differentiate the response in the gross lardaceous type from that in the small atrophic contracted type of amyloid disease, and it is for this reason that I am going to stick to my diagnosis in the face of a 24 per cent removal of Congo red from this patient's blood. If he turns out to have had small kidneys with rather quantitatively small amounts of amyloid, then the test could still be consistent with the clinical evidence.

Dr. Benjamin Castleman: Do you want to comment about the size of the spleen?

Dr. Chapman: I assumed that it was part of the whole process, that is, that he had an amyloid spleen.

Dr. Castleman: Dr. Dameshek, you saw this patient before he entered the hospital. Would you like to add anything?

DR. WILLIAM DAMESHEK: When I first saw him the problem was not so much the urinary one that had been in the background but rather the enlarged spleen and anemia. The anemia was of the macrocytic variety, associated with a slightly elevated white-cell count and the presence in the peripheral blood of myelocytes and nucleated red cells. These findings in association with a large spleen make one think of myeloid metaplasia of the spleen, with a faulty liberation of myelocytes and nucleated red cells into the peripheral blood. I like to consider that myeloid metaplasia is not "agnogenic" or "idiopathic" as some observers believe but that it is secondary to some sort of more fundamental disorder of the bone marrow, which is often sclerotic, such as the condition reported by Dr. Chapman[2] some years ago.

X-ray studies of the bones, which were not done here apparently because the patient was too ill, showed the typical picture of osteosclerotic anemia, with irregular areas of osteoporosis and increased density of the pelvic bones and the femurs. Since a sternal puncture failed to bring up enough marrow material for diagnosis, we then did a sternal trephine bone-marrow biopsy, which showed sclerosis of the bone and bone marrow, but in addition hyperplasia of the white-cell elements, interpreted by a pathologist as leukemia. I did not think that this diagnosis was warranted and was inclined to consider the case as one of osteosclerotic anemia with myeloid metaplasia of the spleen. Later, because the patient was going downhill, we tried the effect of a few x-ray treatments over the spleen. The spleen actually (not "allegedly") went down in size and the patient's red-cell count went up. Theoretically, this should not occur in myeloid metaplasia of the spleen. In the background was the chronic urinary infection, which at the time I studied the patient was minimal. Regarding the matter of amyloid disease, I must admit I gave it no consideration.

DR. WYMAN RICHARDSON: I saw this patient and thought that the blood picture suggested pernicious anemia, complicated by a uremic state, although there was also some immaturity of the cells seen in the blood film. I thought that it might be the result of the complicating uremia. I also suggested that he be given liver extract, on the bare chance that he might respond. This was tried, but he had no response.

CLINICAL DIAGNOSES

Pernicious anemia?
Chronic sepsis.
Uremia.

DR. CHAPMAN'S DIAGNOSES

Chronic bacterial sepsis (scrotum and kidneys).
Amyloidosis of kidney and spleen.

ANATOMICAL DIAGNOSES

Splenitis, acute and chronic, with marked splenomegaly.
Epididymitis and orchitis, acute and chronic.
Pyelonephritis, acute and chronic.
Pulmonary emphysema and congestion.
Cardiac dilatation.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The autopsy on this man showed an enormous, moderately firm spleen; it weighed slightly over 1000 gm. In gross we obviously thought of myelogenous leukemia. In fact there were two small infarcts, which one often sees in myelogenous leukemia. The vertebral and sternal bone marrow on gross examination was red, and the marrow in the tibia and femur, which ordinarily is fatty, was pink, indicating hematopoietic hyperplasia. We were not impressed in the gross with the sclerotic character of the bone marrow.

The scrotum still contained a sinus tract, and there was an acute and chronic epididymitis. The epididymis was filled with pus, and the process had extended into the testis. Both kidneys were pale and slightly enlarged, the combined weight being 400 gm. Microscopically they showed an extreme pyelonephritis that undoubtedly was secondary to the scrotal abscesses, the orchitis and the epididymitis. The ureters were not distended but were moderately injected. There was no evidence of amyloid in the kidney or the spleen.

Microscopical examination of the spleen showed a picture that, had one not known about the history of the case, one would have called subacute bacterial endocarditis. There was edema, and widening of the Billroth cords. In the sinuses and throughout the pulp were plasma cells, lymphocytes, hemosiderin-laden phagocytes and polymorphonuclear cells. The appearance was that of long-standing chronic infection. Certainly it could not be called leukemia. That was borne out by the microscopical appearance of the bone marrow, which showed many mature polymorphonuclear and plasma cells, as well as cells of the red-cell series. There was some fibrosis, but this was slight and merely a secondary manifestation of long-standing hyperplasia of the bone marrow. I do not believe that one can call it an osteosclerotic type of anemia. It is the largest spleen that we have seen in chronic sepsis.

The only additional finding that we would have liked to demonstrate in this case is a bacterial endocarditis, but there was none. The lungs were emphysematous and congested, but we found nothing to explain the recent productive cough. The heart was dilated but not hypertrophied.

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CASE 31232

Presentation of Case

A sixty-seven-year-old woman was admitted to the hospital because of jaundice and drowsiness.

Three or four months before admission she complained of severe pain that appeared to involve the entire epigastrium. A month later she developed a severe nonproductive cough, which was diagnosed as bronchitis. Ten days before admission she became jaundiced and began to suffer from generalized pruritus. The jaundice and pruritus were progressive, although the degree of jaundice seemed to fluctuate. During the few days prior to entry she became increasingly drowsy and unintelligible.

Many years before entry she had had gallstones removed, but the gall bladder was not taken out.

Physical examination revealed an obese, drowsy, deeply jaundiced woman. The skin showed excoriations from scratching. The breath was "mousy." The few remaining teeth were carious. A Grade 2 systolic murmur was heard over the entire precordium. The heart was enlarged, extending by percussion 14 cm. to the left of the midsternum. The abdomen was distended and showed a definite fluid wave with shifting dullness. The liver was palpable three fingerbreadths below the costal margin. There was slight tenderness in the right upper quadrant of the abdomen. A rectal examination was negative. The ankles showed moderate edema.

The temperature, pulse and respirations were normal. The blood pressure was 112 systolic, 60 diastolic.

Examination of the blood showed 15.3 gm. of hemoglobin and a white-cell count of 16,400, with 72 per cent neutrophils, 22 per cent lymphocytes and 6 per cent monocytes. The red cells and platelets were normal. The urine was dark amber, with a specific gravity of 1.018, and gave a +++ test for albumin and a ++++ test for bile. The sediment showed 15 red cells, 75 white cells and occasional granular casts per high-power field. A stool was black and tarry and gave a ++++ guaiac test. The serum nonprotein nitrogen was 50 mg. per 100 cc., the phosphorus 2.8 mg., the icteric index 180, the prothrombin time 44 seconds (normal, 18 to 20 seconds), and the alkaline phosphatase 6.0 Bodansky units. The fasting blood sugar was normal.

The temperature rose to 101.0°F., and the pulse to 100, and the respirations quite abruptly rose on the third day to 35. The patient received intravenous glucose and vitamins, in addition to a high-carbohydrate, low-fat diet given through a nasal tube. She also received a whole-blood transfusion. The stupor deepened, and she quietly expired on the fourth hospital day.

Differential Diagnosis

Dr. Bernard Jacobson: I should like to make a few comments on some of the findings. Three or four months before admission the patient complained of severe pain. I assume that it was not one episode of severe pain but the beginning of recurrent pain of several months' duration. We are not told anything about her appetite or her general sense of well-being, but since she was apparently well nourished when she came in, I assume that she did not lose much weight. We are told that the degree of jaundice seemed to fluctuate, which is of some interest. By and large, severe cases of catarrhal jaundice are apt to have less fluctuation of the degree of jaundice than do those with stone in the common duct or carcinoma of the head of the pancreas.

The breath was "mousy." This of course is of some significance because a mousy odor is due to the presence of certain amines from the liver cells, and that usually signifies diffuse atrophy. But it is fair to say that, although it is suggestive of diffuse atrophy of the liver, it is not pathognomonic. We are told farther on that the liver was palpable three fingerbreadths below the costal margin. In an obese woman of this age that may not mean enlargement at all. It may mean a liver that has been pushed down one or two fingerbreadths and be a perfectly normal-sized liver for such a person.

We are not told anything about other masses in the right upper quadrant. Certainly one would be looking for masses other than the liver. I should be interested to know if there was a mass that corresponded to the size, shape and position of the gall bladder. On the other hand, the fact that none was noted in a woman with an obese abdomen may not be of any significance. A liver palpable three fingerbreadths below the costal margin is certainly against the diagnosis of acute yellow atrophy. The preceding history of the case — jaundice of ten days' duration and rapid progress to death on the fourteenth day — is consistent with the diagnosis of acute yellow atrophy, but the presence of a liver that did not shrink in size is definitely against such a diagnosis. We are told later that the patient had a hemoglobin of 15.3 gm. I rather suspect that this patient's hemoglobin was less; she may have been dehydrated at the time the initial blood count was taken. Nonetheless, that is an important finding, because it rules out for further consideration of such a rare diagnosis as congenital hemolytic jaundice.

Rather upsetting to me, with my preconceived opinion of what this case might have been, is the report that the stool was black and tarry. It seems to me that it would take a lot of blood to color a stool black, especially one that should have been rather light in color because of the absence of much bile pigment. It could not have been due to a recent hemorrhage. That is the first indication that there had been bleeding high in the gastrointestinal tract.

I believe that we can dismiss several of the common causes of jaundice in a person of this age. It certainly was not catarrhal jaundice or diffuse in-

fectious hepatitis. There had been far too much pain, and there had been a rather rapid development of extremely severe jaundice. The course of the disease was quite unlike what one would expect in that condition. I have already mentioned that I do not believe that this was a toxic acute yellow atrophy. We are told that the patient had gallstones removed many years before, which brings up the question, Did this patient have a complication of the previous cholelithiasis? Did a stone, for instance, in the common bile duct cause all the present jaundice? There is nothing in the history that is against that diagnosis or that rules it out. On the other hand, that certainly does not explain some of the present findings — the ascites and the gastrointestinal bleeding. Cirrhosis would not be expected to give this degree of jaundice and to terminate in such a short period, ten or fourteen days.

We now come to the likeliest diagnosis — some form of neoplasm. Certainly in a person with a previous history of cholelithiasis we have learned in recent years to think of carcinoma of the gall bladder or of the bile ducts as a not uncommon cause of death in a person with jaundice. The diagnosis of carcinoma of the gall bladder or bile ducts is sometimes difficult. One cannot always palpate the mass in the gall-bladder region. That was the diagnosis that I favored until I came to the story of bleeding from the upper gastrointestinal tract. I must say that I was disappointed to read about that tarry stool.

Could this have been one of the most frequent causes of jaundice in a person of this age, namely, carcinoma of the head of the pancreas? There is nothing in the entire picture that rules out that diagnosis, except, again, the gastrointestinal bleeding.

Could this have been a primary hepatoma or a diffuse carcinomatosis primary in the liver? It could have been, except for the bleeding in the upper gastrointestinal tract. There is nothing else against it. Even the fact that they did not feel a solitary liver mass does not rule out the diagnosis.

We must bear in mind that about a month before she developed jaundice she had a nonproductive cough, diagnosed as due to bronchitis. Possibly that was indicative of metastases to the lungs. So we must think of some type of neoplasm below the diaphragm, with early metastases to the lungs and later metastases elsewhere. On the basis of statistical frequency, carcinoma of the stomach is the likeliest diagnosis. Certainly it would account for most of the findings — metastases to the lymph nodes compressing the bile ducts, metastases to the liver, spreading to the peritoneum and part of the cause of the accumulation of the fluid in the abdomen, metastases to the lungs, and gastrointestinal bleeding.

As for the terminal episode, I do not know how we can tell whether she had a bronchopneumonia or a terminal pulmonary embolus. We are told nothing about the physical findings at the time. I think that terminal bronchopneumonia is far more frequent in a case like this.

I should say then that this patient had carcinoma of the stomach with metastases to the lymph nodes, compressing the bile ducts, and metastases to the liver, peritoneum and lungs, arteriosclerotic heart disease, passive congestion of the kidneys and terminal bronchopneumonia.

A PHYSICIAN: Do you attach any significance to the elevated prothrombin time in connection with the tarry stool?

DR. JACOBSON: We have no evidence of a bleeding tendency in other parts of the body, but I should guess that bleeding sufficient to have changed the color of the stool from clay to tarry could hardly have been the result of a high prothrombin time.

A PHYSICIAN: What about the possibility of carcinoma of the ampulla of Vater?

DR. JACOBSON: In referring to carcinoma compressing the bile ducts I include the ampulla of Vater or some other place in the biliary tract.

A PHYSICIAN: Would that not explain the tarry stool?

DR. JACOBSON: It might by erosion into the gastrointestinal tract. It is a good possibility.

DR. CHESTER M. JONES: I saw this patient when she was first admitted. She was comatose practically all the time after admission and obviously a hopeless case. I thought at the time that she had malignant disease involving the liver and that it was primary or secondary. I could see no way of establishing a diagnosis.

The stool was a glove specimen, and it was pitch black. It would seem to me that Dr. Jacobson's comment was correct: namely, one would anticipate frank ulceration of the mucosa to underlie bleeding of that extent. On the other hand, with pronounced jaundice of this degree, one often obtains positive guaiac tests in the stools regardless of the underlying cause of the jaundice — whether it is intrahepatic or extrahepatic. This stool was actually tarry. I reasoned as Dr. Jacobson did — that there was ulceration. An ulcer in the presence of a clinical picture like this would make me suspect that there was a new growth that had ulcerated into the stomach or the duodenum. Carcinoma of the ampulla may invade the duodenum with subsequent erosion. I thought that the findings were consistent with spreading neoplastic disease.

The death, I believe, even as it is described here, is not at all suggestive of bronchopneumonia or anything else but complete failure of the liver. This woman had enough liver damage to prevent normal function. It is entirely comparable with the sort of picture seen in terminal acute yellow atrophy. We had an excellent example in a case discussed here a short time ago; the patient had carcinomatosis of the liver and a similar death.

All it implies is that the liver is profoundly damaged and that the patient finally dies of hepatic failure. If one had done blood chemistry determinations one would have found that terminally there was some superimposed renal failure.

Clinical Diagnosis

Carcinoma of head of pancreas.

Dr. Jacobson's Diagnoses

Carcinoma of stomach, with metastases to lymph nodes, compressing bile ducts, and to liver, peritoneum and lungs.
Bronchopneumonia, terminal.
Arteriosclerotic heart disease.
Passive congestion of kidneys.

Anatomical Diagnoses

Hepatoma, with metastases to lungs, adrenal glands and vertebras.
Jaundice.
Ascites.
Obstructive cirrhosis of liver.
Acute pancreatitis.
Cholelithiasis.
Dilatation of common bile duct.

Pathological Discussion

Dr. Benjamin Castleman: The autopsy showed about half a liter of straw-colored fluid in the abdomen. There were no peritoneal implants. The liver was large, weighing 2350 gm. About 90 per cent of the parenchyma of the right lobe and 50 per cent of that of the left lobe were replaced by grayish-white nodules from 0.5 to 10 cm. in diameter. The intervening tissue was green. This gross appearance was consistent with a diffuse metastatic process. We looked carefully throughout the body for a primary source but were unable to find one. The stomach showed no erosions, and there were no gross erosions anywhere in the entire intestinal tract. I cannot explain the tarry stool except on the basis of jaundice. Small nodules, 2 or 3 mm. in diameter, were found in the adrenal glands and in the lungs, and most of the vertebral bone marrow was replaced by tumor. With this degree of bone-marrow metastasis it is surprising that the alkaline phosphatase was not elevated and also that there was no anemia.

The liver parenchyma between the tumor nodules showed bile stasis and biliary obstructive cirrhosis. There were no enlarged lymph nodes compressing the extrahepatic bile ducts, but certainly the intrahepatic ducts were compressed and undoubtedly accounted for the jaundice; they may not, however, have accounted for the cirrhosis. The gall bladder did contain a stone, and the common bile duct was dilated, indicating that stones had been passing down for some time. In the head of the pancreas was an ill-defined 2-cm. area of acute inflammation, with foci of fat necrosis. This is further evidence that the stones had been going down the bile duct. I am just wondering whether some of the symptoms that this patient had, especially the pain, rather than being due to the hepatoma itself, might have been due to stone in the common duct that blocked the pancreatic duct and set off a pancreatitis that smouldered along.

Dr. Francis D. Moore: How big was the stone in relation to the caliber of the cystic duct?

Dr. Castleman: The stone measured about 0.5 cm. in diameter, and the duct measured up to 3 cm. in circumference. At some time, a stone could have become impacted in the ampulla at the entrance to the pancreatic duct, thus starting up an acute pancreatitis. I think that that is the best bet and that the jaundice was a combination of extrahepatic and intrahepatic bile-duct obstruction, although there was no stone in the common bile duct or pancreatic duct at the time of autopsy.

Dr. Jones: It is rather surprising that with so much carcinomatous involvement she did not have a long period of jaundice.

Dr. Castleman: Certainly more than half the liver was involved.

Dr. Jones: It is also interesting that there was a striking family history of carcinoma. Various members of the family had died of carcinoma.

Dr. Milford D. Schulz: Was the spleen normal?

Dr. Castleman: It was small.

Dr. Schulz: And the esophageal veins?

Dr. Castleman: They were not enlarged. There was not much portal obstruction. The tumor had invaded the hepatic veins throughout the liver, which accounts for the metastases to the lungs.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

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Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

INCREASING OPPORTUNITIES FOR WOMEN IN THE MEDICAL SERVICES

The opening at Camp Edwards of a 6000-bed reconditioning hospital for wounded and sick veterans is a reminder that the healing of the wound is only a stage in the long process of treating the disabled. This sequence of events may be dramatically initiated with the administration of plasma; it is not finished until the highest possible degree of function is regained, a hopeful attitude toward life is restored, and a new skill, if necessary, is developed.

The amazingly high percentage of lives of wounded soldiers that are now being saved is a modern miracle of science, but it carries with it a great and continuing responsibility — that of making as many as possible of these bodies useful to their tenants and to mankind, and of reducing to a minimum the permanent human wreckage that war always leaves in its wake.

Certain accessory medical services are largely instrumental in the attainment of such goals, and in these services, women are playing the leading parts. Bulletin 203 of the Women's Bureau, United States Department of Labor, describes the present situation as regards physical and occupational therapy, and the strides that these services have taken as a result of the war.

As the bulletin so aptly phrases it, physical therapy is a relatively new occupation in which an old art is plied — that of treating human ailments with heat, light, water, exercise and other physical means. Even greater progress in the development of this service for the future seems assured by the recent Baruch gift for research and specialized training for physicians in the field of physical medicine.

Physical therapists, more numerous than occupational therapists although still fewer than medical laboratory technicians, numbered before the war some 3100, of whom less than 50 were men. Sixteen approved institutions were then graduating about 150 women a year. Thirty-one institutions, including eight army hospitals, now maintain schools of physical therapy, and graduated over 700 technicians in 1944, representing a considerable increase, although a number still far short of the needs for military, veterans' and rehabilitation services.

The occupational therapist, as distinguished from the physical therapist,

> . . . conducts programs for patients confined in hospitals and other institutions to provide them with directed activity and to assist in their rehabilitation; plans and organizes work projects for patients; supervises workers who teach and direct patients in assigned activities, . . . studies patients' reactions while they are engaged in work situations, and writes reports describing their conditions.

Something under 2000 occupational therapists, registered and unregistered, were practicing by 1940; five approved schools were listed in 1941, a number that had grown to twenty-one in 1944. Ninety-nine per cent of occupational therapists are women.

Although the care of war casualties has, of course, opened up unlimited opportunities for these medical services employing women, they need have no fear of facing any serious postwar depression. The country will need these relatively new professional groups so long as it continues to expect a high grade of medical practice and hospital care.

MEDICAL BIBLIOGRAPHY

Much of modern medicine stems from Sir William Osler, and in the later years of his life, one of his principal interests was medical history and medical bibliography. He stimulated the late Fielding H. Garrison, of the Army Medical Library, to make out a list of the most important contributions to the literature of medicine and its ancillary sciences, to show the significance of individual writings in the history and development of the medical sciences. This list[1] first appeared in 1912, and since it was considered by Garrison "as a convenient scaffolding for a book," he later wrote his famous *An Introduction to the History of Medicine*,[2] a standard work invaluable to all students of the subject. When Garrison moved to Baltimore to become librarian of the Welch Medical Library, he expanded the list and republished it in 1933 under the title "Revised Students' Check-List of Texts Illustrating the History of Medicine, with References for Collateral Reading."[3] In the last ten years, a further revision with additions and annotations was undertaken by Mr. Leslie T. Morton, the librarian of St. Thomas's Hospital Medical School in London. This important and outstanding reference work has now been published.[4]

Garrison's revised check-list contained 4186 entries; Morton has retained 3826 and has added 1680 new bibliographical notes. Some of the additions are to the important contributions made between 1933 and 1943, but many of the other sections have been expanded, with the idea of showing the significance of the entries more clearly. In addition, Morton has provided an author and subject index. The majority of references have been verified, and many minor corrections have been made. In a few instances, owing to the war, it was not possible for Morton to see the original publication, but he has fortunately had the help and advice of friends in checking references that were not available to him in London or in other British library centers.

The list as published by Morton is extraordinarily complete and accurate. Its bibliographical matters, such as the spelling of names, the dates of birth and death of the author, the exact names of secondary contributors and the title and notes on the contents of each paper, sometimes not clear from the title, are features of this work for which only a trained librarian with a bibliographical instinct could be responsible.

The matter of how much to say about each contribution is something most difficult to decide, particularly in relation to a publication of recent date. No one, for instance, would question the propriety of putting in the paper by Lawrence O'Shaughnessy in 1936 on an experimental method of providing a collateral circulation for the heart as an outstanding contribution to medical advancement. That O'Shaughnessy, as stated by Morton, was killed in Flanders in May, 1940, while serving with the R.A.M.C., although important biographically, is of slighter value from the point of view of bibliography. The same might be applied to the note regarding John Locke's "Essay" stating that it occupied him off and on for twenty years. When Morton writes, however, that a book published in 1575 in Spanish, which is difficult to obtain, was translated and issued in 1696 in English, or that a publication of 1690 was reprinted in 1910, he is indeed giving important information and something that every student of medical history will welcome. The matter of inclusion or notation, however, is largely a question of individual preference. Even if an international commission of medical historians should meet, there would, no doubt, be many instances of disagreement.

Morton's list is of such importance that it would seem worth while to go beyond an individual compiler and to suggest further revision under the auspices of a combined British and American medical historical commission. Such an endeavor might well be the concern of the Medical Library Association in America and the National Medical Library in Washington, along with a nucleus of medical librarians in Great Britain and possibly in other

countries. It is hoped, moreover, that after the war Great Britain will see fit to set up a medical library association on the lines similar to the associations so well established in America.

Medical bibliography is greatly indebted to Morton for his splendid publication, but in a field that is continually advancing one can only hope that his book will be considered as a steppingstone toward future publications along the same lines. Indeed, Morton himself expresses a similar opinion when he hopes that his book "may be considered worthy to serve as a starting point for something better, to the construction of which both the specialized knowledge of the medical historians and the bibliographical skill of the librarians can at some future date be devoted."

References

1. Texts illustrating history of medicine in Library of Surgeon-General's Office. *Index-Catalogue of the Library of the Surgeon-General's Office* 2 S., 17:89-178, 1912.
2. Garrison, F. H. *An Introduction to the History of Medicine.* Fourth edition. 996 pp. Philadelphia: W. B. Saunders Company, 1929.
3. *Idem.* Revised students' check-list of texts illustrating history of medicine, with references for collateral reading. *Bull. Inst. Hist. Med.* 1:333-434, 1933.
4. Garrison, F. H., and Morton, L. T. *A Medical Bibliography: A check-list of texts illustrating the history of the medical sciences.* 412 pp. London: Grafton and Company, 1943.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

ANSHIN — Marcus M. Anshin, M.D., of Lynn, died May 21. He was in his fifty-third year.

Dr. Anshin received his degree from Loyola University School of Medicine, Chicago, in 1919 and did postgraduate work in Vienna and Berlin. He was a fellow of the American Medical Association.

His widow, a daughter and a sister survive.

FRASIER — Joseph A. Frasier, M.D., of New Bedford, died May 20. He was in his seventieth year.

Dr. Frasier received his degree from Columbia University College of Physicians and Surgeons in 1896.

His widow, a son and two sisters survive.

HURLBUT — Lt. Robert S. Hurlbut (MC), U.S.N.R., formerly of Cambridge and Marblehead, was killed in action March 26 when the destroyer *Halligan* was sunk off Okinawa. He was in his thirty-fourth year.

Dr. Hurlbut received his degree from Harvard Medical School in 1938 and was permanent secretary of his class. He was called to active duty in July, 1942, after serving two years as a surgical house officer at the Massachusetts General Hospital. Following service at the Chelsea Naval Hospital at the Office of Medical Officer Procurement, First Naval District, and as physician at the Harvard Naval Dispensary, he began sea duty on the *Halligan* in September, 1943. He saw service in the Atlantic and in North Africa, and was in invasion actions for more than a year in the Pacific.

His mother, his widow and four children survive.

LEVIN — Harry M. Levin, M.D., of Malden, a surgeon in the United States Public Health Service Reserve, was killed on January 29 when the ship he was on was torpedoed at Guadalcanal.

Dr. Levin had been on active duty with the United States Coast Guard since February, 1943. He was first assigned to stations on the West Coast and since September, 1944, had been on sea duty in the South Pacific.

His widow and three children survive.

CORRESPONDENCE

POSTWAR POSITIONS FOR ANESTHESIOLOGISTS

To the Editor: The Hospital Section of the Postwar Planning Committee of the New England Society of Anesthesiology would like to hear from physicians who are interested in the peacetime practice of anesthesiology in New England. This information will be made available to persons interested in obtaining the services of an anesthesiologist in private, group or hospital practice in New England. It is thought that the collection and dissemination of this information may facilitate the proper relocation of anesthesiologists now in the service when they return to civilian practice.

The following information is desired from those who are interested: age, medical school, year of graduation, internship (year and hospital), training in anesthesiology (in detail, giving length and type of training and chief of service), activity and specialty after completion of training, military experience in anesthesia, publications, membership in medical societies, present address and permanent address.

This information should be sent to me as chairman of the committee. The other members are Drs. Jacob H. Fine and Sidney C. Wiggin.

Morris J. Nicholson, M.D.
Lahey Clinic
605 Commonwealth Avenue
Boston 15

BOOK REVIEWS

Ourselves Unborn: An embryologist's essay on man. By George W. Corner, M.D. 8°, cloth, 188 pp., with 18 illustrations. New Haven, Connecticut: Yale University Press, 1944. $3.00.

Here is a book that everyone who is interested in life itself will want to read and to own. It is a scholarly, practical and intensely interesting account of the biography of the first few weeks of life. The distinguished author, who is director of the Department of Embryology, Carnegie Institution, Washington, D. C., and professor of embryology at Johns Hopkins University School of Medicine, presents his subject matter in a pleasant and facile style; here and there he infuses his discussion with provocative quotations from the history of embryology, philosophy, poetry and religion. Yale University was indeed fortunate in choosing Dr. Corner to give the Terry Lectures, which constitute this book. In presenting an account of the natural history of the human embryo, he shows how science can contribute to the broadening and purification of religion itself.

The first lecture considers the topic "The Embryo as Germ and as Archive," and the author concludes this lecture with the following paragraph: "Thus to the seeing eye the human embryo from egg to birth is an archive in which is written the evidence of its descent as an animal, a vertebrate, an amniote, a mammal, a primate; and it is an organic germ, in which the gift of life is intrinsically bound up with the necessity of growth and of ineluctable change. This is not only preparation for life; it is life itself." The second lecture is titled "Prenatal Fate and Foreordination." The author presents the problem of embryonic defect and death in the human species and makes it clear that in man, as in other animals, genetic and constitutional factors are operative in some cases, and disturbances of the maternal environment in others, in the production of prenatal damage and loss. The embryologist sees the human egg as endowed with a general property of organic creatures, and he would never have conceived the idea that an immaterial self-conscious existence is entrusted to the uncertain custody of the human egg. The author declares his conviction that the spirit of man — all that makes him more than a beast and carries him onward with hope and sacrifice — comes not as a high-born tenant from afar but as a latent potentiality of the body. It is true to a

certain extent that the fate of the embryo is foreordained. The third lecture deals with the subject "The Generality and the Particularity of Man." The author indicates that the embryologists could hardly fail to support the most orthodox Darwinian–Huxleyan doctrine of the descent of man from an ape-like ancestor, in close relation with the gorilla and the chimpanzee. Readers will be interested in the concept of man's nature as a "Diagram of the Ladder," a point of view closely interwoven with the older thought of Christianity and the concept of the "Diagram of the Fan," which expressed the findings of evolutionary biology.

The reviewer strongly urges every educated person to read this book and thereby add to his intellectual stature.

The Analysis and Interpretation of Symptoms. Edited by Cyril M. MacBryde, M.D. 8°, cloth, 301 pp., illustrated. Philadelphia: J. B. Lippincott Company, 1944. $9.00.

The striking feature about modern medicine is its increasing emphasis on the physiologic processes, and the trend is toward dynamics rather than static description. Physiologic principles are emphasized not only in the treatment and prevention of disease but also in its diagnosis as well. No longer is it stressed that such and such signs spell a certain medical syndrome, but rather one attempts to understand the changes in a particular case that deviate from the normal. With this view in mind, one can evaluate this book, which is a symposium by a group of prominent specialists, who analyze a small group of frequent symptoms and signs. The discussions are logically and well presented, and the conclusions in many of the papers are often summarized in one or two sentences. As in any book, there are statements with which one can take issue.

As a whole this book will prove valuable to those who would keep abreast of modern medicine, with its physiologic trends and its chemical analyses, which are great aids in diagnosis and tend to supplant that nebulous quality — the clinical sense — which will always have its place in medicine.

The Pathogenesis of Tuberculosis. By Arnold R. Rich, M.D. 4°, cloth, 1008 pp., with 89 illustrations, 20 tables and 4 charts. Springfield, Illinois: Charles C Thomas, 1944. $10.50.

This monumental book is the culmination of the many fruitful years of labor that Dr. Rich has devoted to the pathology of tuberculosis. In the preface, the author states that the purpose of the book is to present the basic factors and principles that influence the occurrence of tuberculous infection and determine its progression or arrest. In his own words, the object has been "to determine the influence of each of those factors upon the pathogenesis of the disease and to correlate into a unified whole the basic interpendent, but at present often isolated, facts that have been given to us by bacteriology, immunology, pathology, clinical observation, experimental investigation, epidemiology and genetics."

In the twenty-one chapters, the writer does just that, and after reading through this book one stands both in awe and in amazement at the thoroughness with which this has been accomplished. He draws not only on his own rich experience but also on the vast amount of literature for the purpose of bringing the reader up to date on the best thoughts and opinions in the diversified fields of pathology and immunology in tuberculosis.

The reader will be well rewarded by referring to the chapters on the bacteriology of the tubercle bacillus, with a full discussion of its chemical constituents and the variations in its forms and virulence, as well as its modes of infection. The greater part of the book deals with pathogenicity versus immunology, and the problems of epidemiology and of individual, native and tissue resistance are thoroughly discussed. No clinician can afford to miss the scholarly dissertations on the mechanism of hypersensitivity and on desensitization and its relation to the characteristics of tuberculous lesions and symptoms.

Despite the formidable and intricate discussions, the style is smooth and the subject matter is presented in a clear and orderly manner. The short summaries at the end of each chapter are extremely helpful. The illustrations are adequate and are beautifully reproduced. As a reference book on the pathogenesis of tuberculosis, the reader will find it complete, concise and authoritative. The clinician, pathologist, bacteriologist and public-health worker should find the book useful and also stimulating.

Technic of Electrotherapy and Its Physical and Physiological Basis. By Stafford L. Osborne, M.S., Ph.D., and Harold J. Holmquest, B.S., B.S. (M.E.). 8°, cloth, 780 pp., with 240 illustrations and 72 tables. Springfield, Illinois: Charles C Thomas, 1944. $7.50.

The authors of this book were evidently under the impression that medicine is both a science and an art and that electrotherapy, being a branch of medicine, should be treated with regard to these two forms of culture. Accordingly they do not confine themselves to mere statement of facts but present a scheme of laboratory tests to prove the authenticity of their claims for almost every method of treatment. The system of laboratory experimentation that is outlined in this book has all the earmarks of scientific precision and artistic form.

Although the authors state in the preface, "This text . . . is not to replace the clinical textbooks now available," the reviewer believes that it compares favorably with any one of them. The fact that it constantly stresses the rationale for choosing a certain physical measure in a given case, based on a familiarity with the underlying electrophysics and electrophysiology, tends rather to add than to detract from its clinical value.

Another praiseworthy feature of this work is the arrangement of its subject matter in a manner that will suit two classes of readers — the busy general practitioner and the specialist or teacher in this field. The former may have neither the time nor the desire to trace each case to its fundamentals in physics, chemistry, mathematics and mechanics. For him the essentials are stated with simplicity. On the other hand, the man who takes more than a lukewarm attitude to the subject will find a plentiful supply of data, references, curves, tables, charts, cuts and diagrams to illustrate, clarify and prove the soundness of the statements that are made by the authors. By way of illustration the book contains a table of logarithms and one of trigonometric functions of an angle. These do not concern the average reader of an electrotherapeutic text and are never found in one. On the surface they may seem to be out of place, but in reality they are part and parcel of those portions of the book that were written for the more profound students of electrotherapy.

On the whole this book is quite different from the usual textbooks on the subject, almost a stranger in their midst, but paraphrasing Shakespeare, one might say: "As a stranger bid it welcome."

Functional Disorders of the Foot: Their diagnosis and treatment. By Frank D. Dickson, M.D., and Rex L. Diveley, M.D. Second edition. 8°, cloth, 352 pp., with 202 illustrations. Philadelphia: J. B. Lippincott Company, 1944. $5.00.

In this book the authors have described clearly and concisely the common disabilities of the foot. There is a decided improvement in this second edition both in the arrangement of the material and in the subjects that are considered. The book is excellently illustrated with line drawings and photographs. It is well written, and the suggestions for treatment are in accord with recognized orthopedic practice.

A book of this sort is timely and should help greatly in arousing interest in a large number of disabilities that are now frequently neglected. The classification of functional disabilities is probably too simple for extensive use. One would like to see more emphasis placed on the muscular faults of the foot, which are so commonly encountered in both children's and adult's clinics. The bibliography at the end of the book has not been revised to include many of the newer contributions in medical literature. But these are minor faults. The book can be recommended unreservedly as the most practical treatise on disabilities of the feet in the English language.

Man Does Not Stand Alone. By A. Cressy Morrison. 12°, cloth, 107 pp. New York: Fleming H. Revell Company, 1944. $1.25.

Through the centuries from Galen, Cicero, Paley and others, detailed arguments have been set forth to show the remarkable adaptation of man to nature. Since the beginning of the twentieth century there have been equal efforts to show the adaptation of nature to man. The present book, written by a scientist, purports to bring to the attention of thinking people the facts now available that tend to sustain

this belief in the adaptation of nature to man and further to indicate its purpose. The author has marshaled his facts rather well and has drawn them from the fields of the solar system and of the chemistry of the atmosphere and the ocean, as well as from the fields of evolution and the physiologic adjustment in animals including man.

Emphasis is laid on the points that there is directive purpose lack of everything and that this purpose is being carried out by a supreme being whom we call God. The high quality of science has always been to find a fact, and although science disclaims any desire to search for God, it is nevertheless true that men of the stamp of Copernicus, Darwin and others had deeply religious feelings. This book gives some support to the idea that only deeply religious people of our largely material age are the earnest men of science. It is perhaps in this sense that the book will make a strong appeal to general readers. The reviewer believes that the author offers reasonable testimony in the way of facts for his thesis that man does *not* stand alone — a keen challenge to Julian Huxley's book *Man Stands Alone*.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Endocrinology: A brief review for physicians. 12°, paper, 169 pp. Circular No. 177. *Heart Disease: An elementary reference for physicians.* 12°, paper, 63 pp. Circular No. 176. Published by the Department of Public Health, State of Illinois, Roland R. Cross, M.D., director.

These two elementary manuals on common disorders are published as part of the postgraduate medical education program of the Department of Health of Illinois. The department has felt the need of bringing advances in diagnosis and therapy to practitioners and consequently has adopted the plan of preparing brief summaries in various fields of medicine for distribution to all physicians in Illinois. It is hoped that through this service the busy doctor can keep himself up to date with a minimum of effort and without taking any time away from his practice.

Organic Chemistry. By Louis F. Fieser and Mary Fieser. 8°, cloth, 1091 pp. Boston: D. C. Heath and Company, 1944. $8.00.

This new textbook has been made useful to physicians by the inclusion of a number of chapters dealing with significant applications of organic chemistry to the biologic and medical sciences. The presentation is in a readable narrative style, with an extensive body of factual data included in the form of comprehensive tables and citations under formulas. It is an up-to-date comprehensive authoritative text in its field.

The Sick African: A clinical study. By M. Gelfand, M.B., Ch.B., M.R.C.P., D.M.R., Government Medical Service, Southern Rhodesia, medical officer, Salisbury Native Hospital, physician to the War Emergency Hospital, government pathologist, Pasteur Institute and Public Health Laboratory, Salisbury. With a foreword by Colonel A. P. Martin, O.B.E., M.D., D.P.H., D.T.M. and H., director of medical services, chief health officer and medical director of Southern Rhodesia. 8°, cloth, 373 pp., with 123 illustrations. Cape Town, South Africa: Post Graduate Press, in association with Stewart Printing Co., Ltd., 1944. 25 shillings.

This clinical manual has been written for the members of the medical profession who practice among the African natives. It is hoped that it will also be of assistance to missionaries and native medical orderlies. The descriptions of diseases have not been gone into in great detail, but an endeavor has been made to stress the salient features of the diseases so that the physician will be able to recognize them without difficulty. Many of the clinical manifestations of disease in the native differ from those of the same diseases in the European, and these differences have been discussed in the light of medical men who have practiced among the natives. The civilization and the outlook of the native in no way resemble those of the European. This fact must be taken into account, for it involves an entirely different angle of approach in investigation and treatment. There are also practical difficulties that should be considered, such as the long distances to be covered, the lack of facilities and the insufficiency of funds available for medical purposes. Much of the detail of helminthology, protozoology and parasitology has been purposely omitted. The author has endeavored to show how best to treat the patient in the most practical and simple way, without losing sight of the difficulties of native practice. Each disease has been discussed in a separate chapter without regard to classification. Colonel Martin in his foreword characterizes the manual as an outstanding work that should meet the needs of all workers in the African field of tropical medicine.

Physiology in Health and Disease. By Carl J. Wiggers, M.D., D.Sc., professor of physiology and director of the Physiology Department, Western Reserve University School of Medicine, Cleveland. Fourth edition. 8°, cloth, 1174 pp., with 246 illustrations. Philadelphia: Lea and Febiger, 1944. $10.00.

The fourth edition of this standard textbook, first published in 1934, has been thoroughly revised and brought up to date in accordance with developments resulting from the war. To keep the book within a reasonable size and to include a great amount of new material, many topics and sections have been rearranged, rephrased and condensed or printed in small type. Many portions have been greatly enlarged, and several new chapters have been included. Approximately one third of the text has been completely rewritten; over a thousand new references to pertinent literature have been added, and the number of illustrations has been increased. Particular attention is called to new or expanded portions dealing with war physiology, for example, discussions of nerve regeneration, eye fatigue, color appreciation, somatic and visceral pain, headache, blood and plasma depletion, principles of transfusion, blood substitutes, blood volume, hemorrhage and shock, abnormalities of blood coagulation, artificial respiration, pulmonary edema, dietary deficiencies, undernutrition and starvation, muscular exercise, physical fitness and fatigue, water deprivation, acclimatization and aviation physiology, with particular emphasis on the effects of pressure changes, anoxia, acceleration and the role of proprioceptor, vestibular and ocular reflexes in flying. Altitude-sickness, airsickness and seasickness are likewise discussed.

Dr. Colwell's Daily Log for Physicians: A brief, simple, accurate financial record for the physician's desk. 8°, cloth. Champaign, Illinois: Colwell Publishing Company. $1.00-$5.00.

This standard account book, especially designed for physicians, should meet the needs of most practitioners. It constitutes a complete system of financial records, with the exception of the ledger. Any form of ledger may be used in conjunction with the *Daily Log*. The space provided accommodates an average practice for a calendar year. For physicians with large practices, however, an edition is issued in two volumes, providing double the space for each day.

Theory of Occupational Therapy. By Norah A. Haworth, M.A. (Cantab.), M.R.C.S., L.R.C.P., D.P.M., assistant medical officer, London Passenger Transport Board; and E. Mary Macdonald, principal, Dorset House School of Occupational Therapy, Barnsley Hall Emergency Hospital, Bromsgrove. With a foreword by Sir Robert Stanton Woods, M.D., F.R.C.P., consultant adviser in physical medicine, Ministry of Health, and physician in charge of the Department of Physical Medicine, London Hospital. Second edition. 8°, cloth, 148 pp., with 68 illustrations. London: Baillière, Tindall and Cox, 1944. $2.50.

In the preparation of this second edition of a manual intended for students and nurses, the text has been thoroughly revised. A considerable amount of new material has been added, especially in the chapter on occupational therapy in a general hospital and also in that on the treatment of cardiac cases and arthritis. The first four chapters discuss the treatment of mental disorders, orthopedic and surgical cases, cardiac diseases and tuberculosis. The remaining chapters deal with equipment, records, finance and training. A bibliography of seven pages is appended to the text.

Modern Clinical Syphilology: Diagnosis, treatment and case study. By John H. Stokes, M.D., professor of dermatology and syphilology, School of Medicine and Graduate School of Medicine, University of Pennsylvania, director, Institute for the Control of Syphilis, University of Pennsylvania, and member, Commission on Syphilis and Cognate Diseases, League of Nations Health Organization; Herman Beerman, M.D., Sc.D. (med.), assistant professor of dermatology and syphilology, School of Medicine and Graduate School of Medicine, University of Pennsylvania, assistant director, Institute for the Control of Syphilis, University of Pennsylvania, dermatologist, Philadelphia General Hospital, and dermatologist and syphilologist, Pennsylvania Hospital; and Norman R. Ingraham, Jr., M.D., assistant professor of dermatology and syphilology, School of Medicine, University of Pennsylvania, associate director, Institute for the Control of Syphilis, University of Pennsylvania, chief, Division of Venereal Disease Control, Philadelphia Department of Public Health, and chief, Syphilis Clinic, Philadelphia General Hospital. Third edition. 8°, cloth, 1332 pp., with 911 illustrations. Philadelphia: W. B. Saunders Company, 1944. $10.00.

The first edition of this outstanding work was published in 1926, and the second in 1934. Ten years having elapsed, it was considered advisable, even under wartime conditions, to bring out this third edition. Approximately 65 per cent of the text has been rewritten. The penicillin chapter and the account of syphilis in public health and military medicine are new. The chapter on prenatal, now largely congenital, syphilis, has been expanded and rewritten. The progress of serologic diagnosis has been reflected in extended space devoted to the biologic false-positive reaction. Every important technic of treating syphilis receives full detail and practical discussion. The attempt to write for the student, the practitioner and the specialist simultaneously really telescopes three books into one. In order to keep down the size of the book much use of small type has been found necessary. Likewise it has been found impossible to incorporate full bibliographic references. The book is highly recommended to all those who come in contact with syphilis in their daily work and for all medical libraries.

Soldier to Civilian: Problems of readjustment. By George K. Pratt, M.D., psychiatric examiner, United States Armed Forces, Induction Center, New Haven, Connecticut. With a foreword by George S. Stevenson, M.D., medical director, National Committee for Mental Hygiene. 8°, cloth, 233 pp. New York: Whittlesey House, 1944. $2.50.

The author is a practicing psychiatrist with military experience and service on the staff of the Mental Hygiene Committee. He has made a careful study of the problems of the serviceman's readjustment. This manual has been written for the lay person who will be faced with these problems when the serviceman is discharged from the military service. In simple language, sensible advice and concrete suggestions are given on how to handle such problems as family life, marital relations, community position and occupational adjustment. Particular attention is directed to the man who returns with a physical or psychiatric handicap. An appendix contains the first outline formulated by the newly created National Committee for Service to Veterans, entitled "Community Service for Veterans: A guide to planning and coordination."

Clinical Practice in Infectious Diseases: For students, practitioners and medical officers. By E. H. R. Harries, M.D. (Lond.), F.R.C.P., D.P.H., medical superintendent, North-Eastern Hospital (London County Council), and Milroy Lecturer, Royal College of Physicians; and M. Mitman, M.D. (Lond.), M.R.C.P., D.P.H., D.M.R.E., medical superintendent, River Hospitals. With a foreword by W. Allen Daley, M.D. (Lond.), F.R.C.P., D.P.H., medical officer of health, London County Council. Second edition. 8°, cloth, 570 pp., with 52 illustrations and 26 tables. Edinburgh, Scotland: E. and S. Livingstone, 1944. $6.00.

The text in this second edition of a work primarily intended for students and first published in 1940 has been thoroughly revised and much new material incorporated throughout the book. The size of the book has been increased by over a hundred pages, and selected references to medical literature lacking in the first edition have been appended to each chapter. This manual has considerable value for ready reference, although it is based on English practice.

NOTICES

ANNOUNCEMENTS

Dr. Isadore Green announces the removal of his office from 636 Beacon Street to 483 Beacon Street, Boston.

Dr. Miriam S. Udin announces the removal of her office from 636 Beacon Street to 483 Beacon Street, Boston.

BOSTON DISPENSARY

A clinical staff meeting of the Boston Dispensary will be held at the Pratt Hospital Auditorium on Monday, June 11, at 12:30 p.m. Dr. Chester S. Keefer will speak on the subject "Use of Penicillin." Luncheon will be served at 12 noon.

NEW ENGLAND PEDIATRIC SOCIETY

There will be a meeting of the New England Pediatric Society on Wednesday, June 13.

PROGRAM

12:00–1:00 p.m. Clinicopathological conference, Children's Hospital.

2:00–3:30 p.m. Clinic, Children's Hospital. Dr. Richard M. Smith and associates.

4:00–5:30 p.m. Clinic, Children's Center, 244 Townsend Street, Roxbury. Dr. Marian Putnam and associates.

6:30 p.m. Refreshments, Longwood Towers.

7:30 p.m. Dinner, Longwood Towers.

8:00 p.m. Behavior Problems in Childhood. Dr. Harry Bakwin, associate professor of pediatrics, New York University, New York City.

Members of the medical profession and students are cordially invited to attend the 8:00 p.m. meeting.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, JUNE 14

FRIDAY, JUNE 15

*9:00–10:00 a.m. Medical clinic. Isolation Amphitheater, Children's Hospital.

10:50 a.m. Hemangioma. Dr. G. Schwartz. (Postgraduate clinic in dermatology and syphilology.) Amphitheater, Mallory Building, Boston City Hospital.

*12:00 m. Grand rounds. Children's Medical Service, Ether Dome, Massachusetts General Hospital.

SATURDAY, JUNE 16

*10:00 a.m.–12:00 m. Medical staff rounds. Peter Bent Brigham Hospital.

MONDAY, JUNE 18

*12:00 m.–1:00 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

TUESDAY, JUNE 19

*9:00–10:00 a.m. Medical clinic. Infants' Hospital.

*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

WEDNESDAY, JUNE 20

*12:00 m. Clinicopathological conference. Children's Hospital.

*12:00 m.–1:00 p.m. Clinicopathological conference, Cambridge Hospital.

*Open to the medical profession.

JUNE 11. Boston Dispensary. Notice elsewhere on this page.

JUNE 13. New England Pediatric Society. Notice elsewhere on this page.

JUNE 14–19. American Board of Obstetrics and Gynecology. Page 364, issue of March 22.

JUNE 16. American College of Chest Physicians. Page xvii, issue of May 10.

SEPTEMBER 17. American Public Health Association. Page 752, issue of November 30.